EVALUATION OF CASES OF BENIGN BREAST DISEASES

Dissertation submitted in partial fulfilment of

the requirement for the award of the degree

of M.S.Degree examination

General surgery

Tirunelveli Medical College

Tirunelveli

THE TAMIL NADU

DR. MGR MEDICAL UNIVERSITY

CHENNAI, TAMILNADU

2013

CERTIFICATE

This is to certify that the work entitled “EVALUATION OF CASES OF BENIGN BREAST DISEASES” which is being submitted for M.S. General Surgery, is a bonafide work of Dr. Kavimozhy Ilakkiya.P, Post Graduate student at Department of General Surgery, Tirunelveli Medical College, Tirunelveli.

DEAN

Tirunelveli Medical College

Tirunelveli

CERTIFICATE

Certified that consolidated dissertation "EVALUATION OF CASES OF BENIGN BREAST DISEASES", presented here by Dr. Kavimozhy Ilakkiya .P, is based on bonafide cases investigated and studied the candidate himself in the wards of Tirunelveli medical college, Tirunelveli.

Prof .Dr S Soundararajan

Professor And Head

Department Of General Surgery

Tirunelveli Medical College,

Tirunelveli.

Date :

Dr. S. SOUNDARARAJAN, M.S.
PROFESSOR & H.O.D.
DEPARTMENT OF SURGERY
TIRUNELVELI MEDICAL COLLEGE
TIRUNELVELI - 11

DEPARTMENT OF GENERAL SURGERY

TIRUNELVELI MEDICAL COLLEGE

TIRUNELVELI

CERTIFICATE

Certified that Dr Kavimozhy Ilakkiya .P, has worked on the dissertation "EVALUATION OF CASES OF BENIGN BREAST DISEASES" under my guidance and supervision. The consolidated report presented here is based on bonafide cases treated in Tirunelveli medical college, Tirunelveli. The observations and conclusions made by the candidate are his own and have been verified by me.

Guide:

20/12/12

Dr R. Maheswari MS,

Associate Professor,

Dept of General Surgery,

Tirunelveli Medical College,

Tirunelveli.

CIVIL SURGEON
Tirunelveli Medical College Hospital
TIRUNELVELI-11.

DECLARATION

This is a consolidated report on "EVALUATION OF CASES OF BENIGN BREAST DISEASES" based on patients admitted in Tirunelveli Medical College Hospital, Tirunelveli during the period from February, 2011 to August, 2012.

This is submitted to the Tamilnadu Dr M.G.R. Medical University in partial fulfillment of the rules and regulations for the M.S degree examination in General Surgery.

Tirunelveli Medical College Hospital
Tirunelveli

P. Kavi
Kavimozhy Ilakkiya .P

ETHICS COMMITTEE APPROVAL

ACKNOWLEDGEMENT

I acknowledge my deep sense of gratitude to Dr. Soundararajan, M.S, Professor and Head of the Department of General Surgery, Tirunelveli Medical College, Tirunelveli for his guidance, constructive criticism, and enduring forbearance of my work.

I am thankful to my Chief and my Guide Dr. Maheswari.R, M.S, Associate Professor, Department of General Surgery, Tirunelveli Medical College, Tirunelveli for her inspiring guidance and enthusiasm through the entire period of study.

My heartful and sincere thanks to our assistant professors Dr.KJP.Selvi, M.S, Dr. Sivanupandian M.S, Dr. Anand Shanmugaraj, M.S, for their valuable suggestions and constant supervision during my course of study and preparation of this dissertation.

I am thankful to the Heads of the departments of Pathology, Tirunelveli Medical College, Tirunelveli for the necessary support.

I owe a great debt of gratitude to my friends Dr. Sumathi and Dr. Menander for their support and help throughout my course of study.

I also thank all my postgraduate colleagues who have been a source of constant help in technical aspects and encouragement during my study.

I thank my house surgeons who helped in collection and compilation of the data.

I thank all the patients for their co – operation without which this dissertation would not have been materialised.

P. Kavi

KAVIMOZHY ILAKKIYA. P

CONTENTS

ABBREVIATIONS

ANDI Aberration of normal development and involution

BBD Benign Breast Disease

FA Fibroadenoma

FCD Fibrocystic disease

HRT Hormone replacement therapy

MRI Magnetic Resonance imaging

EGF Epidermal growth factor

TGF Transforming growth factor

FGF Follicular growth factor

TDLU Terminal duct lobular unit

LIST OF FIGURES

LIST OF TABLES

EVALUATION OF CASES OF BENIGN BREAST DISEASES

INTRODUCTION

INTRODUCTION

Breast is an important feature of female anatomy particularly representing feminity. Contemporary concepts of female beauty and feminity necessitates the breast to be esthetically acceptable in all the situations. It not only symbolises feminity, mammary glands are also important for the survival of the newborn and thus of the species and portrays the womanhood.

Any change within the breast that comes into notice in a female has a significant impact on her well being. So, its more of a psychological and emotional concern. Rather than disease per se , its the fear of malignancy makes her more apprehensive and anxious.

Though breast cancer being centered in any case with a breast lump, every physician has to remember that benign lesions are more common than malignancy. Enough attention has not been given for the benign breast disorders as much as it was deserved. We have only few papers, studies discussed about this subject.

Benign tumours of the breast is 4 to 5 times more common than malignancy. Benign breast diseases are common disorder, in up to 30% of women will suffer from benign breast diseases requiring treatment at some time

or the other in their lifetime.[1] Over 80% of the breast problems present as pain and or lumps in the breast.

The ratio of malignant : benign breast diseases (BBD) is approximately 1 : 10 in the west.[2]

No comprehensive Indian stastics are available. But it is suspected that position may be similar to that obtaining in the west. No age is barred. Breast cancer risk increases with age and same follows a reverse trend in BBD which is primarily seen in reproductive age group. Familial incidence of BBD explained possibly related to a shared environment and lifestyle. The BBD is largely hormone induced and diets such as methylxanthines (eg:coffee, tea, cola) are attributed in its causation.

The nomenclature of BBD is very confusing. This is because over the last century a variety of clinicians and pathologists have chosen to describe a mixture of physiological changes according to a variety of clinical, pathological and etiological terminology. As well as leading to confusion, patients were unduly alarmed or overtreated by ascribing a pathological name to a variant of physiological development.

The term benign breast disease has been used as a portmanteau into which all breast disease excluding malignancy have been cast, thereby blurring the distinction between a variety of benign breast conditions. To sort out this

confusion, a new system(aberration of normal development and involution)ANDI has been described by the Cardiff breast clinic.

Breast lump brings about anxiety and worry to the patient, as do tumours of the breast. There are deep psychogenic reasons, which make the thought of loss of the breast terrifying to the average woman. As cancer consciousness has taken deeper root among the patients, as well as in surgeons, all the benign tumours are surgically removed and subjected for histopathological study where unnecessary surgery can be avoided, but surgery should not withheld in apprehensive patients and in those having pre malignant tumours.

As the surgery is not only curative but also safeguard against development of malignancy in them in future. It relieves anxiety and apprehension of the cancer conscious patients. The exact relationship between the benign and malignant tumour is yet to be established.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

HISTORICAL ASPECTS :

The tumours of the breast, with its uncertain cause, has captured the attention of the physicians throughout the ages.

Huang Di, the yellow Emperor in 2698 BCE wrote the Nei Jing, the oldest treatise of medicine, gives the first description of tumours of breast. Imhotep, an Egyptian physician(2650 BCE), designed the first pyramid and deified as god of healing. The early Egyptian documented many cases of breast tumours including abscesses,trauma and infected wounds. The references were acquired by Edwin Smith in 1862 and presented in New York historical society[3]. Writings from India and Assyria that date from the same period also mention breast tumours .

The scientific method and clinical advancement of of medicine is credited to Hippocrates, the Father of medicine (5^{th} century BC) was the first to distinguish benign from malignant breast neoplasm. In his work on "disease of women" descries the origin of the hard tumours of the breast and value of medical, surgical and cautery treatment in the breast tumours and he believed that knife can cure the tumour which is incurable by surgery. The Greek physician and surgeon, Leonoides of Alexandria (180 AD) credite the first

operative treatment for breast tumours and used the sign of the retraction of nipple to differentiate malignant from benign tumours .

Attossa, daughter of cyrus concealed a tumour in her breast for a long time till it grew big and ulcerated. When she was sent to Demoncedus a famous physician, who is said to have cured her breast tumour. This case may be a benign tumour of the breast. The method of treatment is not revealed[27].

The excision of benign tumours of the breast was recommended in a telescoping view where they could change malignant by 'Mercus Aurelins severimens'(1654).

Lorenz Heister (b. 1683) described mastectomy for cystosarcoma. Alfred-Armand-Louis-Marie Velpaeu(b.1795), professor of clinical surgery at Paris, in his Treatise on disease of breast claimed to described benign breast tumour[4]. Sir Astley Cooper published the Illustration of the Diseases of the breast in 1829 clearly differentiated fibroadenoma from chronic cystic mastitis.

De Chauliac emphasized the need for wide excision of breast tumours. Brodie is thought to have provided the first clear description of cystic disease in 1846. Cheatale and Cutler were the first to acknowledge that nodularity of the breast was not necessarily pathologic, but occurred regularly under physiological conditions such as menstruation[7].

Geschictter et al and Patey continued to differentiate among the clinical syndromes, pain, nodularity and cystic diseases[8]. Haagensen in his classic book on breast maintained a distinction between pain, physiological nodularity and gross cystic disease[9]. Birkett (1850), Velpeau (1856), Bryant (1887) published description of benign breast disease and clearly distinguished between cystic disease.

Cheatale 6 and Cutler (1931) were the first to acknowledge that nodularity of the breast was not necessarily pathologic, but occurred regularity under physiological conditions such as menstruation(grosscyst) and what they described as pain syndrome.

R. Egan(1962) described mammographic imaging in which difference between benign and malignant tumours was made without the aid of clinical findings[5]. Various authors like L.E. Hughes, Parks and Sandison in 1960, demonstrated that the changes commonly described as fibroadenosis were widely distributed in patients who had not claimed to be symptomatic or demonstrated overt disease.

Benign proliferation of the breast are often considered as aberrations of normal development and involution. The cyclical changes due to variations in estrogen and progesterone results in increased mitosis around days 22 – 24 of the menstrual cycle but apoptosis restores the balance across the cycle.

A new nomenclature ANDI (Aberrations of normal development and involution) was put forward in a National breast conference in Cardiff in 1982. Hughes et al(1987) proposed ANDI(aberration of normal development and involution) classification and its accepted by a multinational, multidisciplinary working party which is now universally accepted. This concept allows conditions of the breast to be mapped between normality, through benign disorders to benign breast disease[6].

EMBRYOLOGY:

By 5-6 weeks – mammary ridges or milk ridges(milk streak/galactic band) occurs along the line which is stretched from the axilla to the groin.

By 7th week- the galactic band in the region of thorax form the mammary bud and the rest of the portion regresses. The thickening in mammary bud(Milkhill stage), followed by invagination into chest wall mesenchyme(disk stage) with a tridimensional growth(globular stage).

At 10 – 14 weeks of gestation, invasion into chest wall and the ridge gets flattened(cone stage).The cell of mesenchyme differentiate into the smooth muscles of nipple and areola with simultaneous developing epithelial bud(Budding stage). The buds branches and results in the formation of multiple epithelial strips(branching stage).

Almost by 16 weeks, the strips represents the future secretory alveoli. The secondary mammary changes occurs by formation of hair follicle, sebaceous gland and sweat gland elements. Only sweat gland develops fully at this time. The above changes in breast development is independent of hormonal influence.

The placental sex hormones will enter the fetal circulation by 3rd trimester of pregnancy. This acts as a stimulus for the epithelial strips to canalize(canalization stage) and this stage keep happening from 20th to 32nd weeks.

Near term, 15–25 mammary ducts formed with coaelescence of ducts. By 32 – 40 weeks, parenchymal differentiation occurs and lobular alveolar structure contains colostrum(end vesicle stage). All these results in four fold increase in gland mass, nipple areolar complex develops with pigmentation. The mammary tissue secretes colostral milk(witch's milk) once stimulated and expressed in postpartum(4 – 7 days) via nipple in most of the neonates in both sexes.

And this colostral secretion will decline by 3 – 4 weeks period due to involution of the breast which occurs as a result of placental hormones withdrawal. The end vesicles are further canalized and develop into ductal structures by additional growth and branching, during early childhood period.

ANATOMY :

Diseases occurring in the breast are best understood in the context of its normal anatomy. Though the breast is present in both males and females but functionally important in later. Breast is an adapted sweat glands invested within the superficial fascia that divides into two layers. The anterior layer separates the relatively small subcutaneous fat lobules and the larger lobules of mammary fat. The posterior layer of superficial fascia rests on the deep fascia derived from the pectoralis major and serratus anterior. The condensations of fibrous tissue between the two layers of fascia(suspensory ligaments of Cooper) divides the breast into lobes. Mammary glands contains abundance of adipose tissue and dense connective tissue. The breast size is determined by the adipose tissue content within the breast. It extends vertically from 2nd to 6th ribs and horizontally from sternal edge medially and almost to midaxillary line laterally. The breast tissue prolonged towards the axilla as the " tail of spence". The retromammary space is the loose areolar connective tissue that is present between breast and fascia.

NIPPLE AND AREOLA :

It projects centrally placed,conical or flattened and is at the level of the 4th intercostal space. Colour varies from pink to light or dark brown and hairless. It has sweat glands and small sebaceous glands give rise to uneven

surface produce known as Montogmery tubercles. Lactiferous ducts opens into its wrinkled tip of nipple via 15 – 20 minute orifices.

LOBES OF THE BREAST:

Each lactiferous duct opens into nipple, after draining the lobes of the breast. All these lobes are arranged radially.

Lobe contains 20 – 40 lobules

↓

Lobules contain 10 – 100 alveoli (tubulosacular secretory units)

↓

Terminal duct

↓

Segmental duct

↓

Collecting duct(15 – 20)

↓

Opens through separate orifice in nipple

The detailed anatomical knowledge of larger duct system and terminal duct lobular unit(TDLU) helps in the exact pathological location in various disorders of breast.

For example:

a) Larger duct : benign papilloma

b) TDLU: Fibroadenoma in developmental phase

c) Cyst and sclerosing adenosis in involutional phase

d) Intralobular –terminal duct: carcinoma

BLOOD SUPPLY :

The main blood supply is via the second perforating branch of the internal mammary and lateral thoracic branches of the axillary artery.

Arterial supply :

1. From the axillary artery via its lateral thoracic and acromiothoracic branches.

2. From the internal mammary artery via its perforating branches, pierce the first to the fourth intercostal spaces, then traverse the pectoralis major to reach the breast along its medial edge. The first and second perforators are the largest of these branches.

3. From the intercostal arteries via their lateral perforating branches relatively

unimportant source.

Venous drainage:

The entire venous network is devoid of valves in the breast.

The axillary, internal thoracic and the third to fifth intercostal veins drain the mammary gland. Venous system consists of superficial and deep venous plexuses. They anastamose with the interglandular system and in the more superficial plane there are subdermal veins and this special network around areola constitute the “circle of Haller”.From here is formed a plexus with a very wide mesh, the “Subcutaneous plexus of Haller” that inturn drains into the superficial veins of the following regions,

1. Above into superficial cervical plexus (anterior and external jugular veins)
2. Laterally into cephalic vein via the thoracoacromial vein
3. Below into the superficial veins of the abdominal wall notably to superficial thoraco-epigastric by communication with the plexus of the opposite breast.

Deep venous drainage accompanies the arterial flow. Intercostal veins course via azygous and vertebral veins into superior vena cava. The internal thoracic perforators empty into the innominate veins. Pectoral perforators flow into the lateral thoracic vein reaches the axillary vein

A rich subareolar venous plexus drains via the intercostal, internal mammary and axillary veins. The distribution of major lymphatics follows the blood supply.

The importance of venous drainage of breast, is being the fact that lymphatics follow the vascular pathway and make the whole point clear the various mode of metastasis that occurs in breast carcinoma.

LYMPHATIC DRAINAGE:

The lymphatics of the breast are thin-walled, valveless vessels that drain unidirectionally except when obstructed by inflammatory or neoplastic disease. The superficial subareolar lymphatic plexus drains primarily the skin over the breast, nippleareola complex. This plexus is interconnected with the deep lymphatic plexus, which drains most of the breast parenchyma. The flow commences from the superficial to deep plexus and from the subareolar plexus through the lymphatic vessels of the lactiferous duct to the perilobular and deep subcutaneous plexus.

Approximately 3 percent of the lymph from the breast is estimated to flow to the internal mammary chain, whereas 97 percent flows to the axillary nodes.

These axillary nodes are divided into three groups according to their relationship to the pectoralis minor muscle .(Anatomic level)

Level 1, nodes lying below the pectoralis minor

Level 2, nodes lying behind the pectoralis minor

Level 3, nodes lying above the pectoralis minor

Drainage of lymph nodes is sequential from level 1 to levels 2 and 3, and even drainage in retrograde fashion to the subscapular and interpectoral groups of nodes also noted. A small amount of lymph drains from the superior aspect of the breast directly to the apical nodes in level 3, bypassing nodes in levels 1 and 2. About 25% of lymph (mainly from the medial half of the breast) drains to the internal mammary nodes in the second, third and fourth intercostal spaces.

The axillary lymph nodes (20–30) drain not only the lymphatics of the breast but also those of the pectoral region, upper abdominal wall and the upper limb

and are arranged in five groups.

1 Anterior

2 Posterior

3 Lateral

4 Central

5 Apical (through which all the other axillary nodes drain)

Lymphatic capillaries fuse to form channels, finally terminates in thoracic duct in left side and empties into left subclavian vein whereas the right lymphatic duct into right subclavian vein.

Nerve supply:

Nervous supply of breast by the anterior and lateral cutaneous branches of 4^{th} to 6^{th} intercostal nerves which carry sensory and sympathetic efferent fibers. Lateral cutaneous ramus of T4 primarily supplies the nipple. This forms an extensive nerve plexus within the nipple, its sensory fibers terminating close to epithelium as free endings, meissener's corpuscles and merkel disc endings.

PHYSIOLOGY OF BREAST:

Breast structure varies with age, time in menstrual cycle, pregnancy and lactation. It is under the control of neuro-endocrinal system. Hormones such as oestrogen and progesterone play a major role. Three major types of physiological changes are seen in the breast physiology. They are :

a. Growth and involution of the breast related to age
b. Changes that are associated with menstrual cycle
c. Changes that occur due to pregnancy and lactation

Pre-pubertal:

In the neonates there are lactiferous ducts but no alveoli, and until puberty little branching of ducts occur, slight mammary enlargement being due to growth of fibrous

stroma and fat.

During menstrual cycle:

In the follicular phase i.e., day 3 to day 14 the stroma is less dense,changes within the lumen of duct with no secretion. In the luteal phase i.e., day 15 to day 25, increase in the stromal density, the ducts have open lumen containing secretion. Due to prolactin and increased endogenous oestrogen has found to exert histamine like action and thereby increasing microcirculation blood flow, 3 days prior to menstruation.

Due to increasing interlobular edema ,females has fullness of breast(increased breast volume) in premenstrual period. Breast volume reaches its minimum at 5 – 7 days after menstruation.

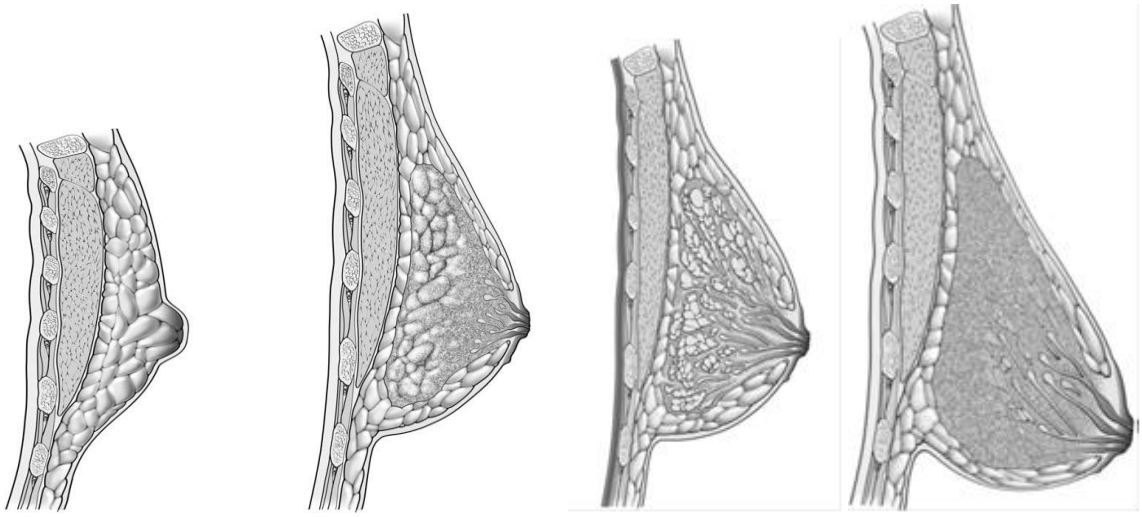


FIG.1: Depicts the changes in breast development

In pregnancy and suckling :

Due to raise in placental estrogen and progesterone, the ducts increase in number and dimension, alveoli proliferate and secretes milk resulting in expansion of alveoli.

Post-lactational breast :

The secretary tissue undergo involution, but the ducts and alveolus never returns completely to pre-pregnant stage.

Menopause :

There will be regression of epithelial structures and stroma. The duct system remains, but the lobules shrink and collapse.

PATHOPHYSIOLOGY :

The aetiology of benign breast disease is not well established, evidence from both animal and human studies is suggestive of a hormonal role in the pathophysiology of benign breast disease. By experiments on animals, development of benign mammary lesions following administraton of oestrogen has been noted. Investigators have found abnormally high oestrogen levels or an imbalance between oestrogen and progesterone levels among women with benign breast disease.

MOLECULAR BIOLOGY :

Normal mammalian breast development depends on a combination of local cell–cell interactions and systemic mammotrophic hormones. The local cellular interactions mediated by growth factors (EGF,TGF-b,FGF), and Wnt gene families.TGF-a both ductal growth and alveolar development. TGF-b may govern early ductal development along with development of alveoli. FGF-1 and FGF-2 are proven angiogenic factors promoting mammary ductal development during sexual maturity. FGF-1 up-regulated in ductal epithelium. FGF-2 is expressed in the mammary stroma. Wnt-1, identified as an oncogene found to be expressed in mammary glands of transgenic mice. The growth factors with regulated expression acts in conjunction with mammotrophic hormone and exhibit mammary differentiation, its growth as well as regression. Further

investigation on this will help us in better understanding of breast development and mammary tumorogenesis.

BENIGN BREAST DISEASES :

Diseases of breast have varied spectrum ranging from deviation from the apparently normal to the serious form such as malignancy. Benign breast diseases have similar presentation beginning from disorder to a well established disease process.

ABERRATION OF NORMAL DEVELOPMENT AND INVOLUTION (ANDI) :

The aberration of normal development & involution(ANDI)classification of BBD comprises the framework for benign conditions of breast that encompasses both pathogenesis & the degree of abnormality.

The ANDI classification was accepted internationally since 1992.

The principles of classification are:

1.Benign disorders are related to the normal processes of reproductive life.

2. The spectrum ranges from normal to aberration to sometimes disease.

3. The distinction between normal and abnormal is pragmatic.

4. The ANDI concept is a unifying concept of symptoms,signs,histology and physiology.

5. The classification is not comprehensive.

It does not include infective/traumatic diseases and male breast pathology is not explicit.

ANDI(ABERRATION OF NORMAL DEVELOPMENT AND INVOLUTION) :

The categorisation of breast disorders has been made more appropriate by relating them to the aberrations of the following normal processes.

1.Development

2.Cyclical change

3.Involution

DISORDER OF DEVELOPMENT

TABLE 1 : DISORDERS OF DEVELOPMENT

Physiological state of breast	Normal event	Benign disorder	Turned out to be a disease
Development	a)nipple eversion b)Lobulardevelopment c)Stromaldevelopment	Nipple inversion Fibroadenoma Adolescent hypertrophy	Subareolar abscess Giant FA Gigantomastia

FIBROADENOMA :

It is considered as an Aberration of normal lobular development. Most important pathological aspect is its epithelial cells arranged in connective tissue stroma. Depending on the stromal component it is classified as

1.Pericanalicular type and

2.Intracanalicular type

Molecular biology studies shows that fibroadenomas are polyclonal in keeping with hyperplasia and phyllodes tumor are monoclonal. They exhibit hormonal influence, sas observed in normal lobules and thus they lactate during pregnancy. During involution, replaced by hyaline connective. Usually,they can

reach upto 3 cm, growth beyond 5cm was very uncommon and regarded as giant fibroadenoma. Fibroadenoma fits well into the ANDI classification.

Small fibroadenoma are " normal"

clinical fibroadenoma(1-3cm) are a "disorder" of normal process.

Giant and multiple fibroadenomas fit in the "disease".

FA commonly occuring in women of age 15 to 25. It is smooth, rounded, firm to hard in consistency and freely mobile within the breast that why it is known as breast mouse and lacks this characteristics features in elderly patients due to involute replaced by fibrosis.

The biological behaviour of FA is variable. Either it may regress, remains the same or grow progressively.

Treatment is by surgical excision of the lump, often removed to alleviate patient concern. If, <25YRS, routine removal will be unnecessary, <35YRS conservative approach is recommended. Cytology is done every 3 months to rule out malignancy. Excision

Of the fibroadenoma is considered after 35 yrs.

FIG.2: Fibroadenoma excision

FIG.3: excised specimen

Studies have shown no malignany risk and supports conservative policy. The occurrence of carcinoma in FA is very rare and to till date only 96 cases totally documented in world literature. If the possibility of carcinoma arises from FA, the epithelial component can be blamed off.

ADOLESCENT HYPERTROPHY:

Breast development associated with gross stromal development. Pathogenesis not known and attributed to the hormonal basis.

An excessively large breast is a disorder. Gigantomastia is the disease end of the spectrum.

DISORDERS OF CYCLICAL CHANGE

TABLE 2 : DISORDER OF CYCLICAL CHANGE

Physiological state of breast	Normal event	Benign disorder	Turned out to be a disease
Cyclical change	Hormonal activity	Mastalgia(cyclical)& nodularity	Severe mastalgia

MASTALGIA AND NODULARITY:

They occur along with normal event such as enlargement of breast during premenstruation phase and postmentrual involution of breast. Painful nodularity that persist more than 1 week of the menstruation is considerd as a disorder. The underlying physiologic abnormality is the increased release of prolactin from pituitary gland by hypothalamic pituitary

axis stimulation[10] and associated with increased caffeine ingestion and ,decresed essential fattyacid intake.

Pathology:

Fibrosis→Cyst formation→Adenosis→Epitheliosis→Papillomatosis→Apocrine metaplasia.

Mastalgia can occur either cyclical or noncyclical and differentiation aided by the use of daily breast pain chart. Cyclical Mastalgia with nodularity also called as mammary dysplasia, fibrocystic disease, schimmelbusch disease, hormonal mastopathy or fibroadenosis.

Females around the age of 30-40 years,esp in nulliparous. Usually bothered by extreme breast discomfort and mastalgia with nodularity in association with menstruation and the same showing waxing and wanning pattern in relation to each menstrual cycle. Prof. Jeffcote has referred to these women as “frustrated multipara” and Hagensen has stressed the psychologic nature of the mastalgia recently.

Diffuse nodular tender swelling, bilateral involvement and predominantly affects the upper outer quadrant with no characteristic mammographic or pathological feature. Changes may resolve with next menstrual cycle.

Aspiration cytology and Mammography are indicated in older women and if doubtful in younger women.

Aim is to exclude malignancy and reassure the patient.

Medical treatment:

1. Evening primrose oil

2. Bromocriptine

3. Tamoxifen

4. Danazol

Various studies has been reviewed on the treatment of mastopathy. The effectiveness of the treatment was more promising with evening primrose oil, bromocriptine, danazol, tamoxifen and low fat diet. The efficacy of norethisterone has yet to be proven. They bring about alteration in metabolism of lipid or serum prolactin. The prolactin acts on lipid metabolism and thereby hypothesised that cyclical mastopathy may be considered as a disorder of lipid metabolism[11]

The effectiveness of treatment with Tamoxifen in mastlagia has been more in favour of non cyclical than cyclical. Its usage in practice to mount its effectiveness in treatment of mastalgia has yet to be established.

DISORDERS OF INVOLUTION

Table 3: DISORDERS OF INVOLUTION

Physiological state of breast	Normal event	Benign disorder	Turned out to be a disease
Involution	a)lobular involution b)ductal involution c)epithelial turn over	a)Macrocysts & sclerosing lesions b)Duct ectasia,nipple retraction c)Mild epithelial hyperplasia	a)cystic disease b)Periductal mastitis/abscess/mammary duct fistula c)epithelial hyperplasia with atypia

CYST FORMATION: ANI(Aberration of normal involution) of a lobule.

They are common in 7% of women in west. The mechanism is unclear. It is explained that involution of a lobular epithelium is dependent on the specialized stroma around it. If stroma involutes too quickly the epithelial acini remain and can form microcysts, precursors of macrocyst formation. One of

such with huge dimensions having a thin capsule-blue domed cyst of Bloodgood.

Cysts originate from Terminal duct lobular unit (TDLU).

Apocrine metaplasia that occurs in the lining epithelium and cyst are almost never malignant.

Types:

1. Apocrine:

The cyst characterised by secretory epithelium(Na : K < 3). They are multiple in numbers(>5). The chance of recurrence is five times higher than other types.

2. Flattened:

They are lined by less active epithelium(Na : K >3). Fluid within the cyst resembles plasma.

3.Mixed type:

Increased incidence between 40-50 years of age and with prolonged use of HRT. Cyst are frequent. Usually single, not uncommon to be multiple and often painful.

The breast cyst are usually solitary.

50% - solitary

30% - 2-5cyst

remaining>5 cysts

Smooth, tense on palpation, mobility not as pronounced as fibroadenoma. Once suspicion of cyst arises, Ultrasonogram of the breast is the investigation of choice and mammography prior to aspiration.

Subclinical cases suggest that they are disorder, not a disease requiring specific treatment.

TREATMENT:

a.Simple aspiration of the cyst

b. Surgical excision-Indication :

1. Blood stained aspirate

2. Recurrent cyst formation

3. residual lump after aspiration

FOLLOW UP :

The suspicion of malignancy arises in following situations:

1. If the cyst is complex
2. If blood stained fluid aspirated from cyst
3. Recurrence of cyst
4. Even after aspiration, either radiologically(mammogram) or clinically evident

   residual within the cyst or with no change in density of the lesion after aspiration.

INTRACYSTIC CARCINOMA ARE RARE (0.1%)

Vargas et al[13] conducted the study to assess the nature and the occurence of malignancy in the clinically detected breast lesions that are cystic in nature. With the aid of ultrasonogram of the affected breast and by image guided biopsy, the study showed that majority of the breast cyst are benign in nature and the occurrence of malignancy was approximately 0.1 percent to 1.2 percent.

Bland[14] and his colleagues in their study observed the role of USG breast is superior to mammography in defining the cystic lesions of breast.

2.DUCT ECTASIA AND PERIDUCTAL MASTITIS :

Bloodgood termed it as “Varicocele tumour” of the breast, 1923.

Fuggier called the condition as “mastitis obliteran”. The principal inflammatory cell present in periductal inflammation was the plasma cell and thereby it was

also been termed as "plasma cell mastitis", and other terminologies were Comedo mastitis, granulomatous mastitis according to its histology. Haagensen was the first to use the term duct ectasia.

Haagensen[15] proposed that the primary event was dilatation of the duct. The secretions stagnate which contains fatty acid, inturn acts as a chemical irritant to the periductal tissue. The process continues to cause the resultant fibrosis, brings about contraction and nipple retraction.

Another theory states the above event in retrograde fashion. The primary event to occur is the Periductal mastitis, can results in damage and disruption of the muscular layer/elastic supporting lamina within the duct and results in secondary dilatation of the duct[16].

In a recent prospective study of 14275 patients determined the inter relationship between Periductal mastitis and duct ectasia. As per the study, both conditions are not a sequential event in either way. They are of separate entities with respect to their etiology and pathology[17]. The age group involved in both conditions does not coincide which complements to the observation made in the study.

Smoking is incriminated as a cause in periductal mastitis and not so in duct ectasia as per the recent studies. Cigarette smoke substances are absorbed orally can damage the duct walls. Organisms such as Staphylococcus aureus or

anaerobic organisms(Bacteriodes and anaerobic streptococci) affects the damaged duct walls and in later date mounts the infection.

Duct ectasia is common among postmenopausal breast and its a part of normal involutional process where as periductal mastitis occurs in a younger age group.

Variable clinical presentation and at the same time they are well recognised. Non cyclical mastalgia, paste- like material discharge per nipple, felt as lump in the subareolar region, retraction of nipple(slit-like) and further complicated by recurrent abscess and fistula formation.

Investigation:

FNAC to confirm the diagnosis. Cytological conformation not always possible.

Treatment:

a. Duct ectasia often improves without treatment.
b. Antibiotic course may be helpful with a symptomatic analgesic treatment.
c. If none of these works , surgical excision of all affected ducts.
d. Abscess can be treated by aspiration , incision and drainage.
e. Smoking cessation.

3.SCLEROSING ADENOSIS:

Disorder of either proliferative or the involutional phase of breast cycle (or both). Proliferation of glandular and stromal element causing enlargement with distortion of lobular unit.

Incidence is at 30-50 yrs of age(child bearing and perimenopausal women). Uncommon cause of a breast lump and raise the suspicion of carcinoma. Smooth,relatively mobile mass, frequently painful. Occasionally it can be a cause of mastalgia rather than mass.

Diagnosis made by histology or mammography. It has similar type of fine stipled calcification as in carcinoma which makes it difficult in differentiating.

Treatment:

Excision to rule out carcinoma.

4. EPITHELIAL HYPERPLASIA:

It is more common in premenopausal period and regress spontaneously after menopause. Disease end of the spectrum is atypical ductal and lobular hyperplasia and involvement of

terminal ductal lobular unit(TDLU) is commonly associated with malignancy.

OLDER CLASSIFICATION OF BENIGN BREAST DISORDERS:

A. INFLAMMATORY CONDITIONS :

Acute abscess

Chronic mastitis

Fat necrosis

Gangrene

Tuberculosis

Filariasis

B. MAMMARY DYSPLASIA :

Cysts

Adenosis

Lobular hyperplasia

Fibrosclerosis

Sclerosing adenosis

C. BENIGN BREAST TUMOURS :

Fibroadenoma

Cystosarcoma phyllodes

Intraductal papilloma

Intraductal hyperplasia

D. BENIGN LESIONS WITH PROBABLE INCREASED RISK OF CANCER :

Gross cystic disease

Papillary apocrine changes

Apocrine metaplasia

Epitheliosis and atypia

Atypical lobular hyperplasia

Multiple intraductal papilloma

CULLIN WERREN CLASSIFICATION[30] :

Benign breast diseases

↙ ↓ ↘

a) Epithelial b) connective tissue c) mixed

↓ ↓ ↓

1)Duct papilloma 1)neurofibroma

1)Fibroadenoma

2)pure adenoma 2)Lipoma 2)cyst adenoma

OTHER BENIGN BREAST DISORDERS:

NIPPLE INVERSION:

Disorder of development of terminal ducts that results in interference with protrusion of ducts and areola and hypothesis proposed for its association with increased incidence of duct ectasia.

Surgical correction not advised in this patients. Reassurance is enough.

MAMMARY DUCT FISTULA : (Zuska disease)

Other infrequent terms by which the above conditions referred is squamous metaplasia of the lactiferous, mammillary fistula. Atkins(1955) was the first person to describe fistulas of the lactiferous ducts but first reported by Zuska(1957) and the first person to recognise the association between fistula and duct ectasia was Sandison nad Wallen.

The sequence of event that occurs as follows :

Nipple inversion→ terminal duct obstruction→ recurrent subareolar abscess→mammary duct fistula.

As per the most recent view, the periductal mastitis/duct ectasia is the cause underlying. Inversion of the nipple has been the most important aetiology for mammary fistula. Patient present to the physician with history of intermittent nipple discharge and also with subareolar mass and characterised by its chronicity.

EPITHELIAL HYPERPLASIA OF PREGNANCY :

The marked hyperplasia of duct epithelium give rise to papillary projection which inturn can give rise to bilateral bloody nipple discharge.

BENIGN DUCT PAPILLOMA :

It is a common condition that occurs as a result of disorder of cyclical epithelial activity. It present as a definite subareolar mass with serous or bloody or sanguinous nipple discharge with short duration of history.

3 TYPES:

1. Solitary intra ductal papilloma

2. Multiple intraductal papilloma

3. Papilloma of nipple :

a) Papillary adenoma (common type)

b) Solitary papilloma

CYSTOSARCOMA PHYLLODES :

Muller coined the term “Cystosarcoma phyllodes”, this tumour is neither cystic nor sarcomatous and thus the designation should be abandoned in favour of the a whitish, fibrous appearance. In Phyllodes , the tumour activity varies from benign to locally aggressive to metastatic tumours.

Similar to FA, phyllodes is a fibro epithelial tumour. Its been differentiated from FA by its unique stroma(cellular and sarcoma like) and also malignant nature is determined by the same stromal cell. Phyllodes are rapidly

growing, non-capsulated but still it is a well circumscribed tumour. They don't invade the skin usually, instead pressure necrosis occurs due to its rapid growth.

FIG.4: Cystosarcoma phyllodes with pressure necrosis

FIG.5: cystosarcoma phyllodes

CLASSIFICATION :

Phyllodes tumour is classified based on Degree of stromal cell atypia, number of mitoses, tumour margin characteristics, abundance of stromal cells as follows.

1. Benign
2. Borderline
3. Malignant

BENIGN PHYLLODES :

Clinically the breast lump appears to have smooth surface and defined borders. Histopathologically characterised by hypocellular stroma with minimal nuclear atypia and low mitotic activity.

Approximately 5 - 25 percent of phyllodes tumors are described as malignant.

Fewer than 20 percent of the malignant tumors metastasize. Metastases usually spread hematogenously to the lungs, pleura, or bone. Axillary lymphadenopathy are rare, if present indicates grave prognosis.

INVESTIGATION :

On mammography, phyllodes tumors appear as lobulated, round, or oval masses. Usually noncalcified and well circumscribed. In ultrasonography, phyllodes tumors appear as well defined, solid masses with heterogeneous internal echoes without posterior acoustic attenuation.

It is often difficult to distinguish between benign and malignant phyllodes tumors on the basis of sonographic or mammographic findings. In such a situation, T2-weighted MR images can be helpful. Phyllodes tumors are usually identified as oval, round or lobulated masses, Circumscribed margins and homogeneous high signal intensity.

FNAC and core biopsy aid in diagnosis. Confirmatory diagnosis greater with histopathological examination of excisional biopsy.

Management :

Histological confirmation is necessary.

Primary excision along with a normal tissue margin of 1cm is the treatment of choice, as the tumour does not have a true capsule.

For local recurrence, re-excision or simple mastectomy can be done.

Poor results are obtained with chemotherapy and radiotherapy for recurrence or metastasis.

Microscopy :

Phyllodes tumour shows hypercellular stroma with pleomorphism where as the giant fibroadenoma shows hypocellular stroma, phylodes tumours are well demarcated but may invade the pseudocapsule. This explains the tendency for local recurrence following simple enucleation.

The following hitological features are in favour of malignant phyllodes :

1. higher mitotic index

2. stromal component proliferation is higher compared to glandular component

3. Cytologic atypia

4. peripheral growth invasiveness with infiltration of the surrounding tissues

ACUTE MASTITIS :

The condition develops when bacteria gain access to the breast tissue through the ducts. Usually develops during the early weeks of nursing. Staphylococcal infections induce single or multiple abscesses accompanied by typical inflammatory changes.

BREAST ABSCESS:

1. Lactating breast abscess
2. Non - lactating breast abscess

1. Lactating breast abscess :

It is caused by Staphylococcus aureus but can also caused by Staphylococcus epidermidis and Streptococci. The infection is attributed to abrasions or cracks of the nipple via which the organism gain entry to cause the disease. In a lactating women, the suckling infant's oral flora gain its entry via crack nipple, multiply in stagnant milk and cause the disease. The mother presents with fever, pain, tenderness, swelling of affected breast. The fluctuation is an unreliable late sign.

FIG.6: Breast abscess

Axillary lymph node enlargement is unusual. Clinically patient will be toxic and biochemically evident with leucocytosis.

Treatment with antibiotics followed by repeated aspiration or incision and drainage of the abscess. Advise mother to stop breast feeding in the affected side and continue with normal breast. The infant is not harmed by certain antibiotics including flucloxacillin, co-amoxiclav and erythromycin, with in the

milk. Antibiotics that are contraindicated during lactation include tetracycline and ciprofloxacin.

FIG.7: Incision & drainage of Breast abscess

Kaur and Minocha V.R.[18] in their review article summarize the treatment of breast abscess. A better personal and infant hygiene and early treatment with antibiotics can prevent occurrence of lactational breast abscess. Small breast abscesses can be managed conservatively, by repeated aspiration with oral antibiotics and large abscesses may necessitate incision and drainage.

FIG.8: placement of drain in breast abscess

NON LACTATING BREAST ABSCESS :

A)Periareolar infection :

Most common in younger and mean age is 32 year. The disease has been preceded by peridutal mastitis. The above condition should not be mistaken for duct ectasia as they are common in postmenopausal women and are of different entity.

B)Peripheral non lactating breast abscess :

They are uncommon when compared to above described condition. Diabetes, rheumatoid arthritis and patient on steroid treatment are more likely to be associated with such type of breast abscess. But many manifested the disease without any prior factor. Most importantly, malignancy underlying has to be ruled out with consideration to her age. Staphylococcus aureus is the causative factor.

Usually seen among premenopusal and postmenopausal women.


FIG.9: periareolar breast abscess

ADENOMAS

Adenomas of the breast are well defined tumors. Histopathologically characterised by sparse epithelial elements with inconspicuous stroma. The latter one help to differentiate from fibroadenomas, as stroma is the major constituent.

Though the condition is more of pathological diagnosis, practically they can be categorised as follows :

1. Tubular adenomas
2. Lactating adenomas

1.Tubular Adenomas :

It occurs commonly in younger age group. Clinically present as a lump in the breast or as a nodule in the breast with well circumscribed borders, firm

consistency and freely mobile and often diagnosis of fibroadenoma is clinically made.

Microscopic examination :

Adenomas separated from the adjacent breast tissue by a psuedocapsule and composed of proliferation of uniform, small tubular structures with a scanty intervening stroma.

2.Lactating Adenomas (Nodular Lactational Hyperplasia)

As the name suggest, the breast disease occurs in relation to pregnancy and lactation. Clinically present as lump in the breast with well defined margin and exhibit lobulation. On palpation, it is soft. Some believe that lactating adenoma develops due to superimposed lactational changes on a pre-existing tubular adenoma

Epitheliosis :

It’s a pathological diagnosis.Epithelial proliferation in small ducts, ductules and lobules.

INVESTIGATIONS:

To increase the positive predictive value, combination of diagnostic tests used. Ultimately the chance of missing truly positive cases can be significantly

minimised. The same concept is applied during the evaluation of cases of breast lump on the basis of triple assessment.

Breast lump assessment necessitate multi-disciplinary team approach of clinician, radiologist & pathologist and technical staff too.

The role of combined assessment in cases of BBD, is to exclude cancer, to make a definitive diagnosis of benign conditions, to detect clinicaly impalpable lesion, image guided intervention and to consider conservative management in deserved cases thereby avoiding unwanted surgical modality of treatment and for follow up.

Triple assessment include

1. Clinical examination of the patient
2. mammography and/or ultrasound of breast
3. FNAC/Histopathological diagnosis

Mammography:

Mammography either confirm or denies the clinical diagnosis. The suspected lesion is visualised its presence can be demonstrated. A positive predictivity of this imaging modality is 95 percent. Its role is utilised in critical situations where the physician is in doubt of his clinical diagnosis in differentiating the benign from cancerous lesions of the breast.[29]

Mammography signs of BBD:

Primary signs :

1. the outline of the lesion being smooth
2. the shape of the lesion being either ovoid/lobulation/defined circumference
3. homogenous lesion with hypodensity / transradiant lesions
4. calcification that are coarse and smooth

Secondary signs :

1. Transient fat halo
2. Displacement of surrounding breast parenchyma
3. Multiplicity and laterality(bilateral) of the lesion
4. Normal blood flow within the lesion
5. Dimension of radiologically assessed > clinically measured lesion

Even some of the benign disorder can mimic carcinoma due to ‘distortion of the architecture’ of the breast that include biopsy scar, radical scar, sclerosing adenosis and fat necrosis. Microcalcification can be present in microcyst, papilloma, sclerosing adenosis, epithelial hyperplasia with fine ones and in fibroadenoma with coarse calcifications.

Ultrasound :

The investigation can distinguish between a solid and cystic lesion.

FNAC :

FNAC is a quick and cost effective investigation which gives a preliminary histological lead, based on which one can plan either an open biopsy or medical line of treatment.

The major advantage of FNAC with quick reporting helps the physician for immediate alleviation of the patients fear and avoidance of unnecessary surgery in patients with benign breast disease while at same time detecting all patients with breast carcinoma.

Open biopsy :

It is used to both confirm the diagnosis and also for therapy in small lesions(benign).

APPLICATION OF PATHOLOGICAL CLASSIFICATION IN BENIGN BREAST DISEASES :

In a patient point of view, once benign lump is removed, the next concern for her will be for fear of recurrence or chance of developing cancer breast in near future. For a surgeon, the histopathological report help to determine the confirmation of diagnosis, completeness of the treatment undertaken, of

paramount importance is to ascertain the risk of malignancy in near future. As a result , further follow up and evaluation with relevant investigation if needed can be planned well of.

The lack of consensus between pathologist with respect to various microscopic features is important, as they are ascertained in determining the cancer risk in feature note. The initial and the only study undertaken to solve this issue by Page and co-workers and proposed the more practically applicable pathological classification of BBD, forms the basis for consensus statement of American cancer society.

Cancer risk is defined as a liability to develop breast cancer in the ensuing 10 – 20 years, compared with developed in age matched women who have had no breast biopsy.

The following pathological classification help us in ascertaining the risk :

1. There is no risk attributed for adenosis(scleroisng or florid), apocrine metaplasia, macro and/or micro cysts, duct ectasia,fibroadenoma, fibrosis, hyperplasia(miid 2 – 4 epithelial cells), mastitis, periductal mastitis, squamous metaplasia. The above disorders are classified as non proliferative lesions.

2. Moderate hyperplasia(florid, solid or papillary) and papilloma with a fibrovascular core ascertained to have slightly increased risk of about 1.5 – 2

times. They are characterised by proliferative disease without atypia and absolute life time risk is approximately 5 – 7 percent

Its been believed that family history of carcinoma breast in a first degree relative will boost up the risk of cancer espcially in women with proliferative lesions without atypia. But there is no evidence to support the above issue.

3.The atypical ductal and lobular hyperplasia has been ascertained to have moderately increased risk of about 5 times. This lesions are characterised by proliferative disease with atypia. Irrespective of the disease per se to laterality, both breast are at risk in their life time risk is 13 – 17percent.

Page et al[16] reported that the risk of cancer attributed by a positive family history in cases of atypical hyperplasia is increased by two times than estimated.

Breast hyperplasia without a family Cancer Detection Demonstration Project (BCDDP) study also observed similar results.

4. To assign risk, insufficient data proposed for solitary papilloma or lactiferous sinus and Radical scar lesion.

Lynn C.Hartmann et al stratified the increased risk associated with proliferative lesion with atypia and final results have revealed that family history is an independent risk factor[19].

HRT increases the risk of breast cancer development but not so observed in womens with proliferative breast disease with or without atypia.

STANDARDISATION OF HISTOLOGIC CRITERIA :

Ultimately the benign breast diseases are diagnosed by the pathologist and that's been the final. To differentiate Benign lesion from carcinoma in situ in breast, even the experienced pathologist cannot reproduce the results consistently. To sort out this problem, many studies were undertaken. One such study suggest that for diagnosing proliferative breast lesions, a standard histological criteria among pathologists is necessary thereby reducing the inter observer variability. By reducing the inter observer variability, could aid in a consistent result.

FUTUROLOGY:

Active effort is also being made to identify the biological markers/genetic markers from benign breast biopsies. The limitations of morphology was considered in this subject and studies are in the vogue to identify the role of biological or genetic markers especially its application in differentiating atypical ductal hyperplasia and low-grade DCIS. Unfortunately, they share a common immunophenotypes. Cyclin D1, messenger RNA, is the one and only marker found helpful in differentiate the DCIS(low grade) from ductal hyperplasia with atypia.

Markers under study in this regard, includes estrogen receptor, angiogenesis, p53 expression, and HER-2/neu expression. In a recent study it has been observed that irrespective of the status of hyperplasia association with atypia in a benign breast biopsies, its association with angiogenesis has found to increase the cancer risk. Similarly, risk of breast cancer has been increased in a benign breast tissue containing p53 protein accumulation as per current study.

To conclude, the role of biological and molecular markers in ascertaining the cancer risk in a cases of benign breast diseases has not completely established and still considered as an area of active investigation.

AIMS AND OBJECTIVES

AIMS AND OBJECTIVES

RATIONALE BEHIND THE STUDY:

Benign breast disease which cause a great deal of discomfort(both physical and mental) to the patient, who have doubt that it could be malignant, which has to be ruled out. The need for study is to analyse the spectrum of benign breast disease with respect to age, sex, mode of presentation, clinical features and management.

AIMS & OBJECTIVES :

1. To study the patients presenting with various types of benign breast diseases.

2. To study the association of benign breast disease with menstrual status, marital status, parity, family history & personal history.

3. To study the common presentation of benign breast diseases.

4. To correlate the results of FNAC with that of histopathology in all the post operative cases.

5. To study the treatment modalities in benign breast diseases.

MATERIALS AND METHODS

MATERIALS AND METHODS

It is a prospective study aimed at evaluation of cases of benign breast diseases in Tirunelveli medical college & hospital from the period of feb 2011-july 2012 over a period of 18 month.

SOURCE OF DATA:

TIRUNELVELI MEDICAL COLLEGE,HOSPITAL,TIRUNELVELI

DESIGN OF STUDY:

Prospective study

PERIOD OF STUDY :

18 MONTHS

SAMPLE SIZE :

109 cases of benign breast diseases proven histopathologically.

METHOD OF COLLECTION OF DATA :

Study group comprises of 109 female patients with benign breast disease admitted in Tirunelveli medical college and hospital during the period of 18 months. Patients clinically diagnosed as having BBD are admitted for further evaluation. After obtaining an informed written consent from the patient

, detailed history and clinical examination will be done as per my proforma designed. The proforma designed in the questionarre form. With the aid of routine investigations and investigations specific to the disease per se are also supplemented to confirm the diagnosis. The management will be decided according to the provisional diagnosis made after the investigations. The histopathological report followed up. Finally, histopathologically proven cases of BBD will be included in the study with consideration to inclusion and exclusion criteria. If needed secondary analysis of medical records of these patient will be used to fulfil the missed and remaining data. Pathology report of all these patient collected from my collobarating department. The proformas are completed and finally compiled data are to be profiled.

INCLUSION CRITERIA :

1. Females > 12 years

2. Patients admitted with a provisional diagnosis of benign breast disease.

3. Patients who are found to have benign breast tumors on clinico-pathological examination.

EXCLUSION CRITERIA :

1. Females < 12 years

2. Patients diagnosed to have malignancy during evaluation

3. Pregnant females

4. Males

COLLOBRATING DEPARTMENT :

DEPT OF PATHOLOGY

STASTICAL METHOD:

The data represented in tables, bar charts, graphs.

RESULTS OF THE STUDY

RESULTS OF THE STUDY

The present study includes proven cases of 109 patients with benign breast disease in Tirunelveli medical college and hospital over a period of 18 months and the data are profiled.

A) DISTRIBUTION OF BENIGN BREAST DISEASES IN THE STUDY GROUP

TABLE 4 : Distribution of BBD in the study group

Disease	Cases	%
fibroadenoma	72	66.1
FCD	11	10.1
Phyllodes	15	13.8
Tubular Adenoma	5	4.6
Breast Abscess	5	4.6
Epitheliosis	1	0.9
Total	109	100.0

In the study group, among the cases of BBD, Fibroadenoma was more prevalent that accounts for about 72 cases(66%) which is followed by phyllodes occurring in 15 patients(13.8%), fibrocystic diseases in about 11

cases(10.1%), 10 cases of tubular adenoma & breast abscess with equal sharing,1 case of epitheliosis was recorded.

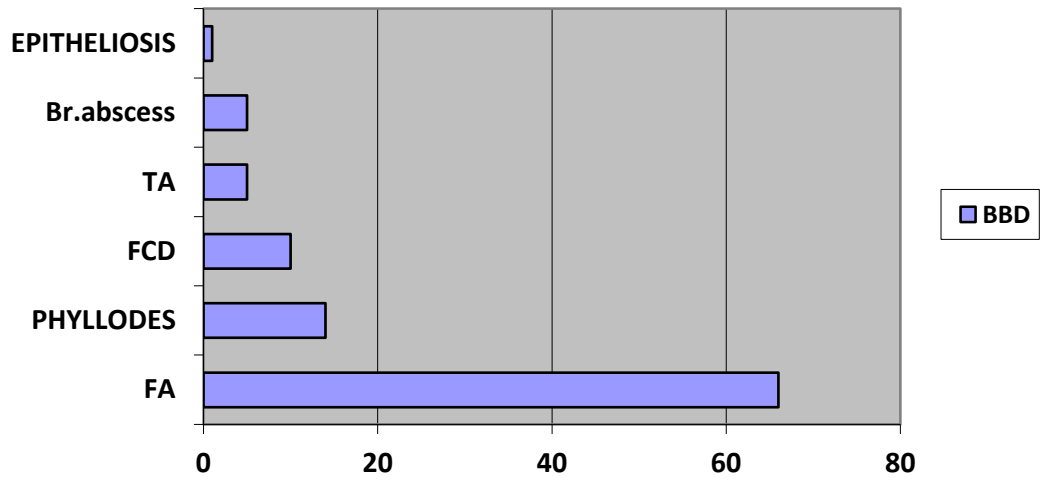


FIG.10: Distribution of BBD

Out of 72 cases of fibroadenoma, 8 cases were giant fibroadenoma.

B) DISTRIBUTION OF BBD AMONG VARIOUS AGE GROUP

FIG.11: Age wise distribution of BBD

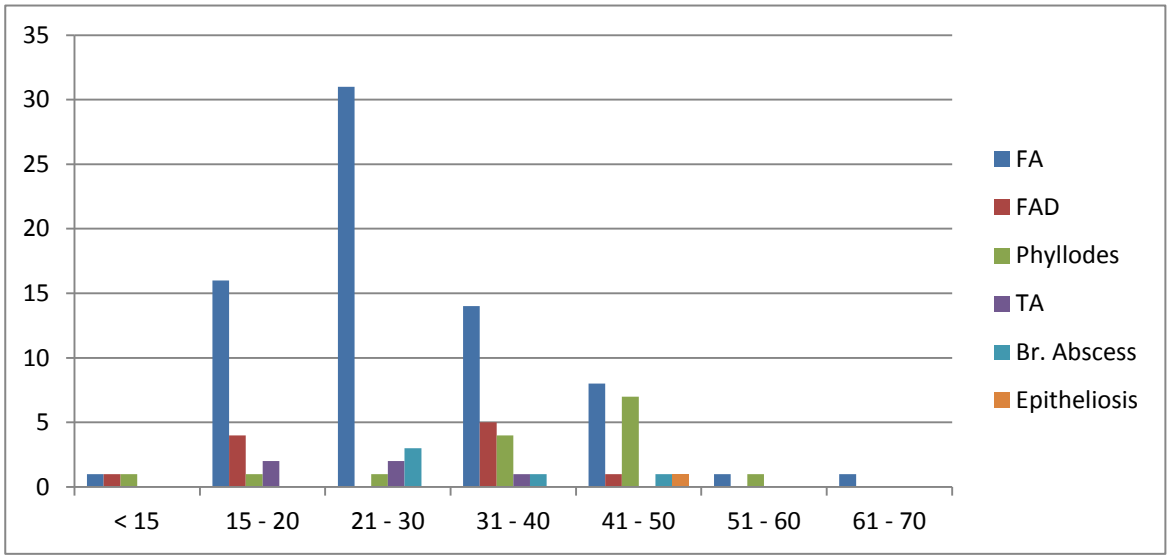
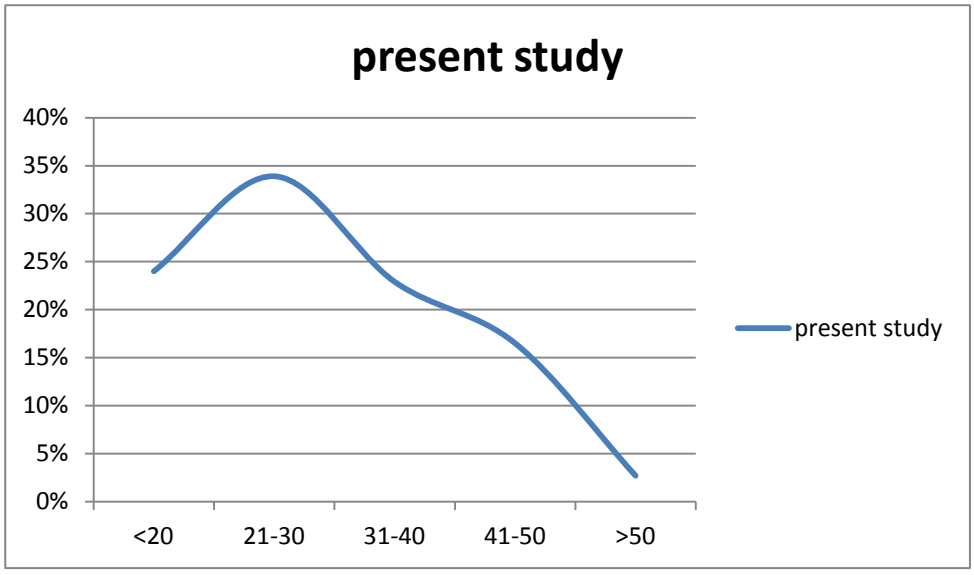


FIG.12: Graphical representation of age distribution



C) MODE OF PRESENTATIONS IN THE STUDY GROUP

TABLE 5: Various modes of presentations of BBD in study group

Chief Complaints	Total	%
Lump	51	46.73
Lump + Pain	58	53.22
Total	109	100

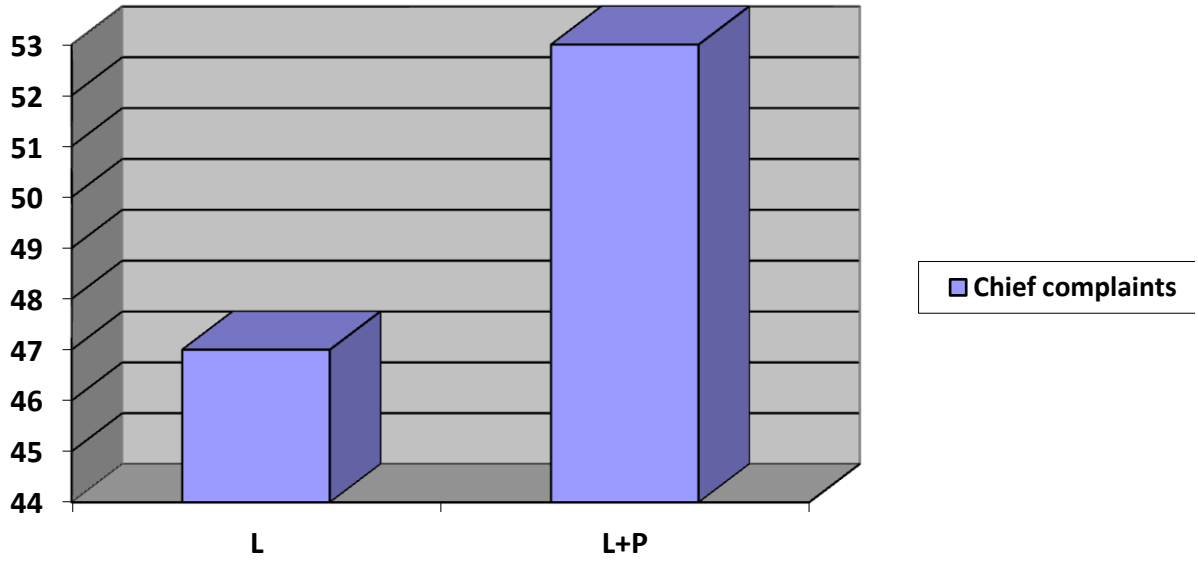


FIG.13: Chief complaints in study group

In study group, majority complained of lump association with pain were 58 cases and 51 cases with lump alone. Among the present study, painless lump being a chief complaint in most of the patients with fibroadenoma & in tubular

adenoma whereas lump associated with pain is been the predominant complaint among the FCD & phyllodes patient in study group.

D) DURATION OF SYMPTOMS

TABLE 6: Study of Duration of symptoms

Month	FA	FAD	Phyllodes	TA	Br. Abscess	Epitheliosis	Total	%
< 1 month	7	2	3	4	5	0	21	19.3
1 - 6 month	51	6	7	0	0	1	65	59.6
7 - 12 month	9	2	4	1	0	0	16	14.7
> 12 months	5	1	1	0	0	0	7	6.4
Total	72	11	15	5	5	1	109	100.0

In the present study,duration of symptoms studied in which most of cases presented witth duration ranging between 1 – 6 months(59.6%).

Out of 65 cases(59.6%) almost majority presented between 1 – 6 months with 51 cases comprised by fibroadenoma patients,9 cases between 7 – 12 months,7 cases in a period of less than a month of the same in the study group.

Out of 21 cases(19.3%),2 cases among FAD, 3 cases among phyllodes,4 cases among tubular adenoma and all 5 cases of breast abscess presented in a period of less than a month.

E) FAMILY HISTORY:

Family history of breast lesion in 6 cases of fibroadenoma was obtained.Out of them. 5 cases gave family history in their siblings and one case in her mother.

F) PAST HISTORY:

4 cases of fibroadenoma had history of operated for breast lump in their past and all these cases had their prior lump only in their contralateral breast.

G) RECURRENCE :

In the present study, 2 cases of recurrence BBD observed .One case of fibroadenoma and other was a phyllodes.

H) MENSTRUAL HISTORY :

Menstrual irregularity were present only in few cases . 6 cases of postmenopausal women had BBD. One case of phyllodes was observed in a female patient(13 years) who has not attained her menarche.

I) PARITY :

Most of the benign breast disease occur after second child birth

J)OCP :

In the present study, OCP usage noted in 7 cases of BBD.

K) MENOPAUSE:

6 CASES observed.

L) DIET :

In the present study, majority were consuming mixed diet(94.5%) and vegetarian accounts for 5.5% of BBD.

M) LATERALITY IN THE STUDY GROUP :

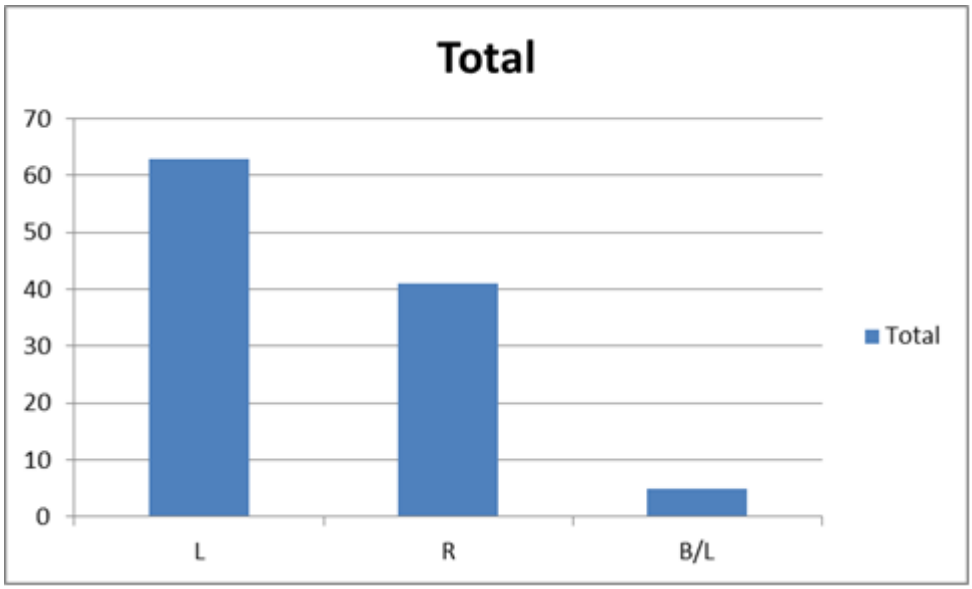
TABLE 7: Laterality among study group

Side	Total	%
L	63	57.8
R	41	37.6
B/L	5	4.6
Total	109	100.0

In the study group,most of the cases occurred on left breast,63 cases(57.8%) than right breast with 41 cases(37.6%) recorded. Bilateral involvement noted in 5 cases(4.6%).

Unilateral involvement of BBD is noted with few cases of bilateral involvement also observed.

FIG.14: Laterality of BBD in study group

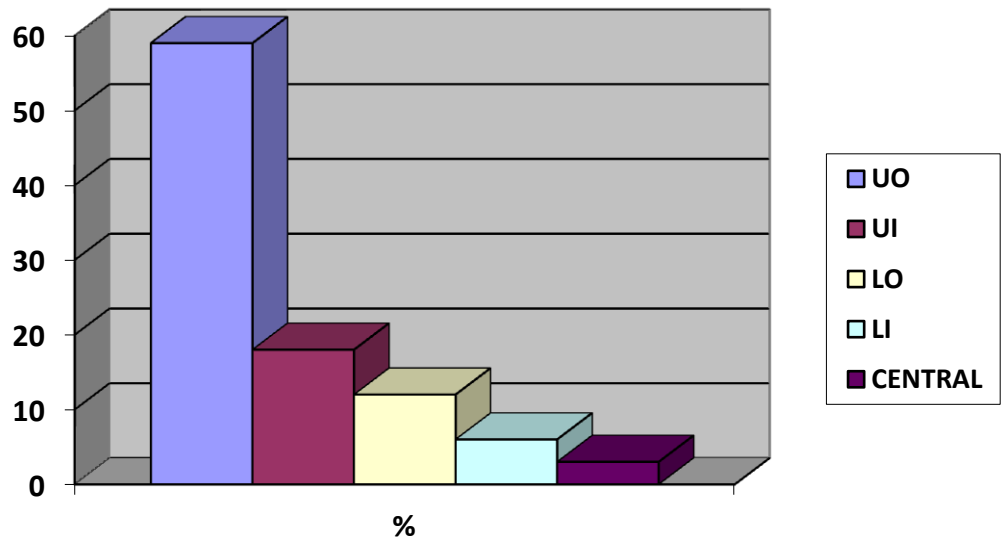


J) QUADRANTS INVOLVED

TABLE 8 : Study of quadrant Involvement in BBD

Quadrant	Frequency	%
UO	64	58.7
UI	20	18.34
LO	14	12.84
LI	7	6
Central	4	3
Total	109	100.0

FIG.15: Distribution of BBD among quadrant



In the present study its observed, BBD predominantly occurred in upper outer quadrant in 64 cases(58.7%), followed by 20 cases(18.34%) occurred in upper inner quadrant, 14 case(12.84%) in lower outer quadrant, 7 cases noted in lower inner and 4 cases in central quadrant.

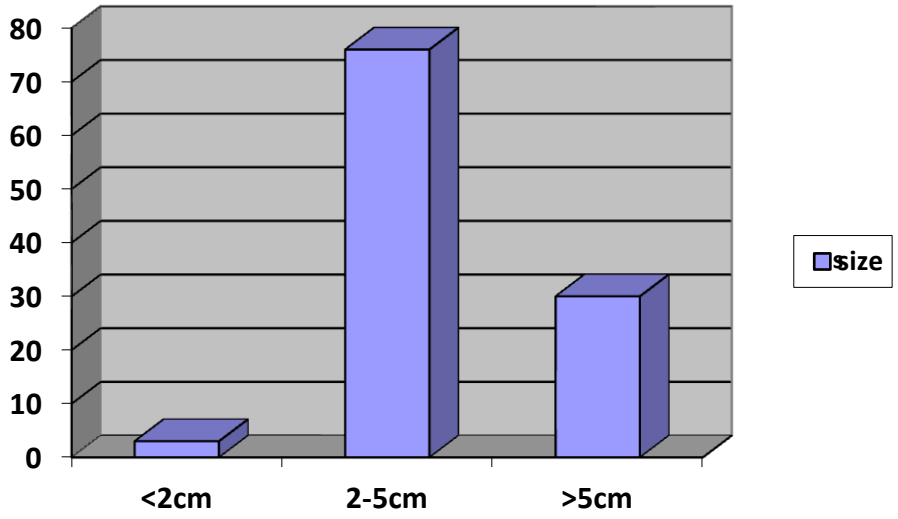
In the study BBD involved predominantly the upper outer quadrant,considerable no of cases noted followed by upper inner> lower outer>lower inner >central quadrant of breast.

K) STUDY OF DIMENSIONS IN THE STUDY GROUP

Table.8: Study of dimension of lesions

DISEASE	**<2CMS**	**2-5CMS**	**>5CMS**	**TOTAL**
FA	2	58	12	72
FCD	-	10	1	11
PHYLLODES	-	3	12	15
TA	1	3	1	5
BR. ABSCESS	-	1	4	5
EPITHELIOSIS	-	1	0	1
TOTAL	3	76	30	109

FIG.16: Dimensions in BBD



L) CONSITENCY & MOBILITY:

Most of the cases were in firm in consisteny with varying mobility demonstrated.

M) SENSITIVITY AND SPECIFICITY OF DIAGNOSIS:

CLINICOPATHOLOGICAL CORRELATION :

TABLE 9: COMPARISON OF CLINICAL DIAGNOSIS WITH H/P/E IN FIBROADENOMA

CLINICAL DIAGNOSIS	**FIBROADENOMA AS H/P/E**	**FIBROADENOMA AS NOT H/P/E**	**TOTAL**
POSITIVE	67	22	89
NEGATIVE	5	15	20
TOTAL	72	37	109

Sensitivity : 93.05%

Specifity :40.54%

Positive predictive value : 75.2%

Negative predictive value : 75%

In cases of fibrocystic disease, the sensitivity and specifity of the clinical examination are as follows:

Sensitivity : 45.45%

Specificity : 4.5%

CYTOHISTOPATHOLOGICAL CORRELATION IN CASES OF FIBROADENOMA :

Table 10: COMPARISON OF FNAC IN CONSISTENT H/P/E

FNAC	**CONSISTENT WITH FA AS H/P/E**	**INCONSISTENT WITH FA AS H/P/E**	**TOTAL**
POSITIVE	65	23	88
NEGATIVE	4	17	21
TOTAL	69	23	109

Sensitivity : 93.05%

Specifity :40.54%

Positive predictive value : 75.2%,

Negative predictive value : 75%.

N) TREATMENT OF BBD IN THE STUDY GROUP

Table.11: REPRESENTATION OF TREATMENT OF BBD IN THE STUDY GROUP

DISEASE	**EXCISION**	**WLE**	**MASTECTOMY**	**I&D**
FA	72	-	-	-
FCD	11	-	-	-
PHYLLODES	9	5	1	-
BREAST ABSCESS	-	-	-	5
TUBULAR ADENOMA	4	1	-	-
EPITHELIOSIS	1	-	-	-
TOTAL	97	6	1	5=109

HISTOPATHOLOGY :

1) Out of 15 cases of phyllodes,

Benign – 2

Borderline - 1 (recurrent case)

Low grade – 9

Intermediate grade – 2

High grade -2

2) Fibroadenoma with focal duct epitheliosis, noted in 3 cases.

3) Fibroadenoma with FCD was reported in a case of 39 years female.

4) One interesting case of benign phyllodes recorded in a 13 year female who has not attained menarche. Provisional diagnosis of fibroadenoma was made and FNAC supported the same. Excision of the lump on H/P/E reported as benign phyllodes.

DISCUSSION

DISCUSSION

The present study was conducted among 109 female patients of BBD in TVMCH from feb 2011 – july 2012 in order to profile the pattern of BBD in all its comparable aspects. All available literature on BBD were reviewed in detail in study group and the following observations were made.

Table 12: Comparison of present study of BBD

DISORDER	**PRESENT STUDY**	**SANDHYA P.IYER(21)**	**RANGABHASHYAM BASHYAM**	**SUSHILA KHANNA(20)**
FIBROADENOMA	66.1%	35%	56.7%	40.8%
FIBROCYSTIC DISEASE	10.1	28.3%	14.2%	13.8%
BREAST ABSCESS	5%	15%	7.9%	-
MASTITIS		8.3%	2.7%	-
OTHER MASS	6%	5%		
PHYLLODES	13.8%		2.3%	13.8%

In study group, fibroadenoma was the most common BBD observed(66.1%). Our observation from the study was consistency was with the authours too. The next common disease was phyllodes followed by Fibroadenosis with Fibrocystic disease .

A retrospective study of 22 years stated that fibroadenoma of the breast was the commonest lesion followed by cystosarcoma phyllodes and fibrocystic disease is consistent with study group.[22]

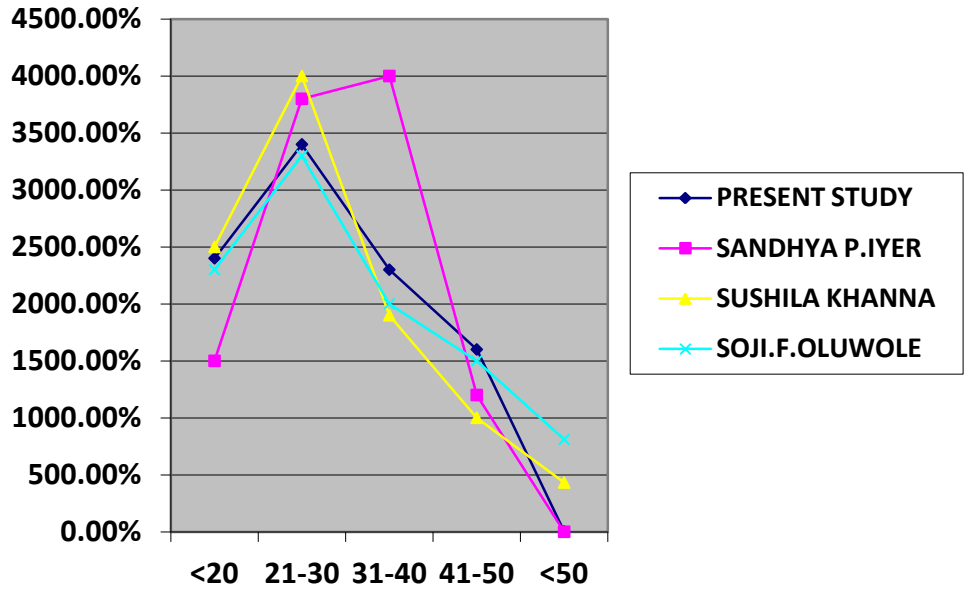
Cases of Tubular Adenoma and Breast abscess has also been reported in study group .

Table13: COMPARISION OFAGE GROUPS INVOLVED IN BBD

AGE	**PRESENT STUDY**	**SANDHYA P.IYER[21](%)**	**SUSHILA KHANNA[20](%)**	**SOJI.F.OLUWOLE STUDY[23](%)**
<20	23.9%	15.2	25.28	23.13
21-30	33.9%	38	40.06	33.13
31-40	22.9%	35	19.56	20
41-50	16.5%	11.8	9.98	15.62
>50	2.7%	-	4.32	8.13

The single best indicator of the probable underlying pathology of a breast mass or breast lump is the age of the patient. Most of the patients were in active reproductive years(20 -40 years) and the finding coincides with the occurrence of aberrations in most active years of womens life. Occurence of BBD is also observed in extremes of age thereby no age is immune to BBD.

FIG.17: Comparison of age group in BBD



In study group, the youngest patient was 13 years and the eldest was 65 yrs. In Indian study on BBD at Varanasi[23], the maximum incidence was in the age group of 21 – 30 years(43%). This finding correlates with the present study.

Fibroadenomas mostly observed in earlier age group whereas FCD are predominantly observed among 20 – 40 year age group and it depicts the fact they are variants of repeated cyclical changes in the form of menstruation, pregnancy and lactation.

Table 14: COMPARISON OF COMMON PRESENTATION OF BBD

In study group, the most common presentation as lump associated with pain is observed in 63.8% respectively. In the study compared, the most common presentation was painless lump was the major complaint.

COMPLAINTS	PRESENT STUDY(%)	S.WILKINSON & A.P.M. FORREST(%)
LUMP	46	71
LUMP+PAIN	53	14

FIG.18: Comparison of common presentations in BBD

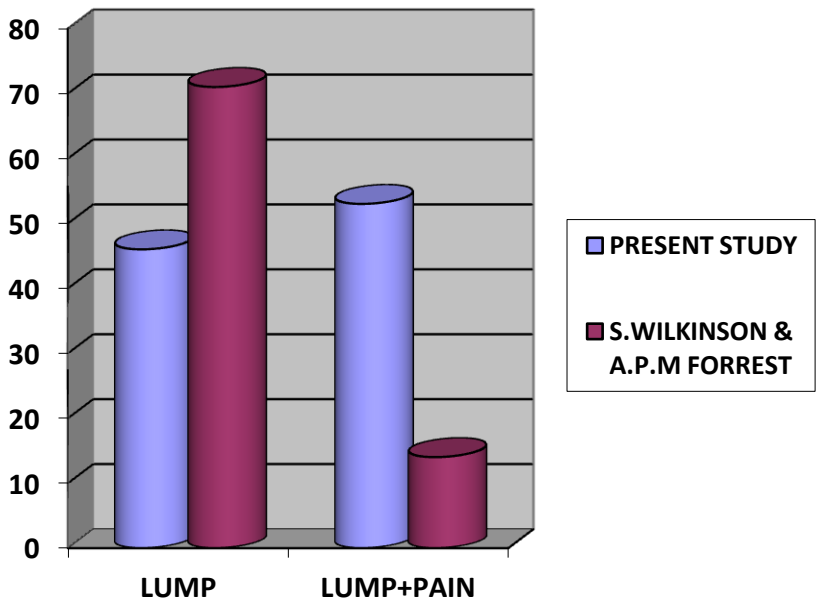


Table 15: COMPARISON OF DURATION OF SYMPTOMS

DURATION	**PRESENT STUDY**
<1 MONTH	19.3%
1-6 MONTHS	59.6%
7-12 MONTHS	14.7%
>1YEAR	6.4%

In the study group, the common presentation of symptoms within 6 months. These patients who presented earlier helped us not only in treating them at early stage of the disease, but also to alleviate the patient fear to exclude cancer.

FAMILY HISTORY & OCP :

Role of both were controversial in literature in causation of BBD. Study group does not support the influence in causation.

PAST HISTORY :

4 Cases had past history of lump breast operated in contralateral side in cases of FA.

MENSTRUAL HISTORY :

The relation of irregularity seen in few cases, whether this carries any significance is inconclusive.

PARITY AND LACTATION :

When breast has understandably gone through the cycle of menstruation, pregnancy and lactation repeatedly after second child birth where BBD is common.

In lactating females, the most common lesions include breast abscess because act of suckling introduces infections.

MENOPAUSE :

In the study group, menopause is not an exception to BBD (6 cases).

DIET HISTORY :

Majority of the cases were consuming mixed diet in study group that probably support the role of diet in occurrence of BBD in the present study.

LATERALITY :

Table 16: COMPARISON OF DISTRIBUTION AMONG SIDE

SIDE	**PRESENT STUDY(%)**	**SOJI.F.OLUWOLE STUDY(%)[23]**	**ETIM.E.ONUKAK (%)[25]**
RIGHT	37.6	45	43.8
LEFT	57.8	41	48
B/L	4.6	14	8.2

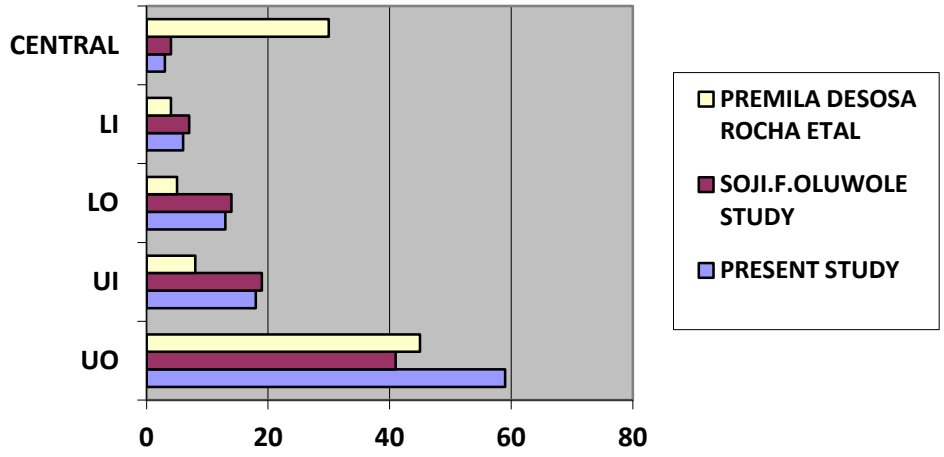
In the present study, left side was the commonest side involvement. The side of breast involved showed no significant difference between either right or left,but commonly lesion involvement is unilateral rather than categorising to side.Bilateral invovement were reported in 5 cases.(25)

QUADRANT :

TABLE 17: COMPARISON OF QUADRANTS INVOLVED IN BBD

QUADRANT	**PRESENT STUDY(%)**	**SOJI.F.OLUWOLE STUDY(%)[23]**	**PREMILA DESOSA ROCHA ETAL 1997[26]**
UO	58.71	41	45.2
UI	18.34	19	7.6
LO	12.84	14	5.2
LI	6%	7	4.4
CENTRAL	3%	4	30.2

FIG.19: Comparison of BBD among quadrant



In the present study,the most common involved was upper and outer quadrant of the breast. This is also in consistent among the Soji F. Oluwole[23] & Premila Desouza Rocha et al study[26]. It is been explained that the proportion of breast tissue in respective quadrants coincides with the occurrence of breast diseases which was proposed in malignancy may also be applied to benign disease also.

SIZE OF THE LESIONS :

In the present study,more number of lesion varied from 2 – 5 cm(69.72%) constituting its average size with overall size ranges from 2 – 10 cm. In Soji.F.Oluwole(23) study more number of lesions varied from 1cm to 10cms. 80% of the fibroadenoma size ranged from 2 -5 cm,corresponds to what is described in literature.

Usually, the largest lesion in size was clinically diagnosed as phyllodes tumour.

CONSISTENCY AND MOBILITY :

As per study group, most of the BBD are firm. Classical mobility in cases of fibroadenoma demonstrated only in 1/5th of the cases.

PROVISIONAL DIAGNOSIS :

When the size of the lesion is more, clinical diagnosis was favoured towards phyllodes and most of them found to be giant FA of breast and vice versa too.

SENSITIVITY & SPECIFITY :

Clinico pathological correlation in cases of fibroadenoma showed 93.5% sensitivity, 40.54% specificity, PPV – 75.2% and NPV - 75 %.

Cytohistopathological correlation in cases of FA showed 94.2% sensitivity, 42.5% specificity, PPV- 5.79 %, NPV – 57.5 %.

TREATMENT :

All patients in study group were managed surgically.

In study group, Excision biopsy was the treatment provided for all cases of fibroadenoma , fibrocystic disease, tubular adenomas. Depending upon size and preoperative investigations, phyllodes tumour treated by wide local excision and

mastectomy for a case of recurrent phyllodes. All our cases of breast abscess were treated by incision & drainage procedure.

Though in some patients of BBD, conservative treatment advised, removal of disease was wanted by the patient for social reason & fear.

The positive predictive value of diagnostic method can be increased with triple assessment to increase the accuracy so that a more truly positive cases would be brought into light and a more appropriate management can be ensued.

CONCLUSION

CONCLUSION

Fibroadenoma is the commonest BBD followed by phyllodes, Fibrocystic diseases of the breast. Most common BBD in Indian subcontinent includes fibroadenoma followed by fibrocystic disease. Benign breast disease commonly occurs during the 2nd and 3rd decade and fibroadenoma being the common BBD in the same age group. FCD commonly seen among 3rd decade and phyllodes commonly observed among 4th decade. It has been noted that no age is immune to BBD. Most BBD observed after second child birth. During lactation, breast abscess cases noted. Role of family history and OCP s in causation of BBD is not significant in the study group.

Most common presentation of BBD was lump in association pain.

Most of them presented with short duration of complaint. So the early presentation could partially be due to the greater awareness of the disease of the breast and fear that breast lump could be malignant.

Unilateral involvement of breast lesion was common, mostly involved the upper outer quadrant and usually a solitary lesion.

Average size of the lesion was about 2 – 5 cm. Most of them were firm with classical mobility demonstrable in fibroadenoma cases, only in 1/3rd of the cases.

All patients in study group were managed surgically. Though the present trend of conservative management has reduced number of surgical procedures. However in view of the anxiety regarding symptoms, distance to be travelled, poor socioeconomic conditions leading to difficulty in follow up, so patients opt for an early surgical method of resolution of symptoms and fear of malignancy. Final diagnosis was relayed upon pathological examination of the specimen and further follow up of the patient planned accordingly.

The sensitivity and specificity of clinical examination was 93.5% and 40.54% and that of FNAC was 94.2% and 42.5% in cases of FA.

One interesting case of benign phyllodes recorded in a 13 year female who has not attained menarche.

SUMMARY

SUMMARY

Most common BBD was Fibroadenoma and predominantly affecting the 2nd and 3rd decade of life. Specific age of occurrence among the various BBDs is noted and also coincides with cyclical changes of menstruation, parity, lactation and involution, as an aberration. Lump associated with pain is the most common presenting complaint. Majority of BBD presented after second parity and infection cases presented in relation to lactation. The role of family history and OCP usage is still to be settled or in dispute. BBD favoured unilateral involvement and majority affecting the upper outer quadrant.

All cases of BBD in the study was managed surgically. The specificity of clinical examination in diagnosing various BBD is fairly accurate. This explains the role of Triple assessment to increase the positive predictive value.

Because of our cultural places importance on breast and fuels a pervasive fear of breast cancer, individuals with breast masses spark strong concern. Patient benefit from early consultation with physician to address their concerns. Management of BBD is an important component in clinical practice. Self esteem is often much improved after surgery. Women should still be encouraged to represent to the service if any new mass appear in future.

" Breast awareness is a goal of breast health movement ."

REFERENCES

REFERENCES

(1)Bailey, love's. Short practice of surgery. Editors, RCG Russell, Norman S. Williams and Christopher JK Balstrode, Arnold Publications, Ch55. page 824

(2) Mansel E. Robert, Fenn J. Nell, Davies L. Eleri, "Benign breast disease and its management ", Chapter 5, recent advances in surgery, No. 21, Johnson C.D., Taylor I., Churchill Livingstone, Edinburgh, 1998 : 71-73 pp..{introduction}

(3) Breasted JH: The Edwin Smith surgical papyrus.Classics of med lib, vol 3,Chicago,1930,university of Chicago press

(4)velpeau AALM: Traite des Maladies du Sein et de la Region Mammaire,Paris, 1854, V Masson.

(5) Egan RL: Experience with mammography in a tumour institution,Radiology 25:894, 1960.

(6) Hughes LE, Mansel RE, Webster DJT. Abberation of normal development and involution (ANDI):A new perspective in pathogenesis and nomenclature of benign breast disorders. The Lancet 1987;1316-1319

(7) Frykberg Eric R., Bland Kirby I., "Evolution of surgical principles and techniques forthemanagment of breast cancer". Chapter 39, The breast – Comprehensive management of benign and malignant disease, 2 nd Edn, Vol. 2 Bland Kirby I., Copeland III Edward M., W.B. Saunders Company,

Philadelphia, 1998 ; 766-801 pp.)

(8) Geschickter. L, Disease of the breast : 2 nd ed. Philadelphia : JB Lippincott, 1945

(9) Mansel, R.E. and Bundred, N.J. (1996) Colour of Atlas of Breast diseases.Mosby-Wolfe,London.

(10) reference:Kumar S et al: prediction of response to endocrine therapy in pronounced cyclical mastalgia, using dynamic tests of prolactin release,Clin Endocrinol 23:699,1985.

(11) Goodwin P.J, M. Neelam, N.F. Boyd. "Cyclical mastopathy : a critical review of therapy". British Journal of Surgery ; 75 : 837 – 844.)

(12) Goodwin P.J, M. Neelam, N.F. Boyd. "Cyclical mastopathy : a critical review

of therapy". British Journal of Surgery ; 75 : 837 – 844.)

(13)Vargas HI, Vargas MP, Gonzalez KD, Eldrageely K, Khalkhali I. Outcomes of sonography – based management of breast cyst. Am J Surg 2004; 188:443-447.

(14)Bland KI: Copeland, Edward M.; The Breast: Comprehensive Management of Benign and Malignant diseases ; vol 2nd Edn. Philadelphia, WBSaunders Company 1998.

(15)) Haagensen CD, Disease of the breast. Philadelphia : WB Saunders, 1971.

(16) page DL.,Anderson TJ: MISCELLANEOUS,non neoplastic conditions.in diagnostic histopathology of the breast,EDINBURGH,1987,Churchill Livingstone]

(17) Scholefield JH, Ducan JL, Rogers K. Review of a hospital experience of breast abscesses. Br J Surg 1987; 74: 469 - 470

(18) Kaur. M, V.R. Minocha. 1999 “The current treatment of breast abscesses”. Indian Journal of Surgery ; 61 : 165-169.)

(19) Lynn C.Hartmann et al,N Engl J Med 2005: 353:229-237,July 21, 2005

(20) Sushila khanna N.C.Arya and N.n.khanna. 1998 “Spectrum of BBD”.Indian Journal of Surgery ; 50 : 169 – 175

(21)Sandhya P Iyer, MA Gore, Epidemiology of benign breast disease in females of child bearing age group, 2000 Jan

(22) Fechner RE : Fibroadenoma and related lesions. In : Page DL, Anderson TJ (eds) : Diagnostic histopathology of the breast, New York, Churchill

Livingstone, 1987 : 72-85 pp)

(23) { Soji .F. Oluwole, Freeman H.P;Analysis of benign breast lesion in Black ; Am J Surg , 1979 : 137}

(24) Wilkinson S. Forrest A .P.M ;Fibroadenoma of Breast ; Br J Surg 1985; 72 ; 838- 40

(25) Onukak E.E, Cederiquist RA , BBD in western population;part 3, BBD in Northern Nigeria;World J Surg 1989 : 13,750-752

(26) Rocha PDS ,Nadkarni NS and Menezes S.; Fine needle aspiration biopsy and breast lesions and histopathologic correlation ; A Analysis of 837 cases in four years;Acta cytol 1997 ; 41(3): 705-712

(27) Bailey, love’s. Short practice of surgery. Editors, RCG Russell, Norman S. Williams and Christopher JK Bulstrode, Arnold Publications,24th Edn, Chapter55,page 824 – 846.

(28)Bland and Copeland. The Breast,comprehensive management of benign and malignant disorder. Editors, Davidson, Page, Recht, Urist, 3rd Edn.

(29) Egas RL: Experience with mammography in a humour institution of Radiology: 1980; 75:894

(30) Lumley JSP Hamilton bailey physical science; Oxford Butterworth – Heinemann 18th Edn 1997

(31) Bland KI, Beenken S.W, Edward M; "The breast" Schwartz's principles of surgery;8th Edn; New York. McGraw Hill 2005 453-499

(31) Jay R.Harris S, Helmen S.Breast diseases 3rd Edn JB Lippincott

(32) Singh IG .Pal G.P- Human embryology, 7th Edn, Delhi, Macmillan 2001, 107-109

(33) Harris, J.R., Lippman,M.,Morrow M. and Hellman, S. (2000) Diaeses of the breast.Lippincott,Philadelphia.

(34) Hughes, L.E.., Mansel, R.E. and Webster, D.J.T. (1989) Benign Disorders and Diseases of the Breast, 2nd edn.Bailliere Tindall, London

(35) Dixon, M. and Sainsbury, R. (1998) Diseases of the breast, 2nd edn Churchill Livingstone, Edinburgh

(36) Oxford Textbook of Surgery (3-Volume Set) 2nd edition (January 15, 2000*):* by Peter J. Morris (Editor), William C. Wood (Editor)

(37) Essentials of Surgery: Scientific Principles and Practice 2nd edition (January 15, 1997): by Lazar J., Md. Greenfield (Editor), Michael W. Mulholland (Editor),

Keith T. Oldham (Editor), Gerald B. Zelenock (Editor), Keith D. Lillimoe (Editor), Keit Oldham By Lippincott Williams & Wilkins Publishers

(38) Ader DN, Shriver LTC**.** Update on clinical and research issues in cyclical mastalgia. The Breast Journal 1998; **4**: 24–32. [An important review on the modern approach towards breast pain integtrating research results with pharmacologic therapies.]

PROFORMA

Name: I.P.no:

Age: unit :

Sex: D.O.A:

Religion D.O.S:

Occupation D.O.D:

Residence

Chief complaints:

Breast lump:side

duration

pain

Nipple changes:discharge/recent changes/indrawn

Any other swelling

Past history:H/Osimilar swelling

History of drug intake:drug details:

duration

Menstrual history:age of menarche:

cycles:

menopause:

mastslgia

Marital history:married/unmarried

Past obstetric history;no of children:

age at each childbirth:

Family history:H/Obreast swelling in other family members:

General physical examination:

Vital signs:

Local examination:

inspection:symptomatic breast examination:

In sitting posture,arms by the side of the body

nipple:position/size/shape/surface/discharge/indrawn/retraction

areola:

skin over the breast:

Arms raised above the head:

Movement of breast:

Leaning forward:

Palpation: side

quadrant:

size:

shape:

surface/margin:

consistency:

Skin fixity:

Fixity to underlying structure:

Examination of lymph node:axillary

supraclavicular

Examination of opposite breast and axilla:

Systemic examination:CVS

RS

ABDOMEN

PROVISIONAL DIAGNOSIS:

INVESTIGATION:

URINE A/S/D:

CBC:

BL.S ,U- ,Cr- .

Blood grouping n typing:

LFT:

Chest X ray: USG:abdomen n pelvis

FNAC

BIOPSY

FINAL DIAGNOSIS:

TREATMENT:

Surgery details:incision:

procedure:

post op:

MASTER CHART

KEYWORDS

U - unmarried

L - lump

L+P - lump+pain

M - month

D - days

R - recurrence

\+ pasthistory

R - regular menstruation

IR - irregular

EN/EX - enucleation/excision

LG/IG/HG - lowgrade/intermediate/highgrade

R - right

L - left

U0 - upper outer

LO - lower outer

UI - upper inner

LI - lower inner

C - central

S.I	NAME	AG	IP.N	MARITA	COMPLAIN	SYPM. DURATIO	MENARCHE AG	REGULARIT	PARIT	LACTATIO	MENOPAU	OCP USAG	NON/VEG DIE	RECURRAN	PAST/FAMI	CLINICAL DX	SI	QUADRAN	CONSISTEN	SIZE(CI	MOBILITY&	NIPPL	FNAC	EX	FA
1	Brindha	23	52992	U	L	1M	12	RMP	-	-	-	-	N	-	-	FA	L	UO	F	2	M	-	FA	EN	FA
2	Fathimuthu Nisha	20	24511	U	L	6M	12	RMP	-	-	-	-	N	-	-	FA	L	UO	F	3	M	-	FA	EN	FA
3	Madathiammal	23	59353	M	L+P	2M	11	RMP	-	-	-	-	N	-	-	FA	B/L	UI	F	3	M	-	FA	EN	FA
4	Shakthi	25	37891	M	L	M2	14	RMP	P2L2	-	-	-	N	-	-	FA	L	UO	F	2	M	-	FA	EN	FA
5	Padma	32	8403	M	L+P	7M	11	RMP	P2L2	-	-	-	N	-	-	FAD	R	UO	F	3	IM	-	FCD	EX	FCD
6	Lakshmi	42	11694	M	L+P	2M	15	RMP	P3L3	-	-	-	N	-	-	FA	L	LO	F	2	IM	-	HYPO CELI	EX	FCD
7	Subbulakshmi	28	51607	M	L	3M	11	RMP	P2L2	-	-	-	V	-	-	GFA	L	UO/UI	F	8	M	+	FA	EN	GFA
8	Parvathy	28	34486	M	L+P	8M	13	RMP	P0L0	-	-	-	N	-	-	FA	R	UI	F	4	M	-	FA	EN	FA
9	Anjali	13	5785	U	L	1M	11	IMP	-	-	-	-	N	-	-	FA	R	UO/LO	F	8	M	+	FA	EN	FA
10	Malarvizhi	17	47305	U	L	2M	13	RMP	-	-	-	-	N	-	-	FA	L	UI	F	4	M	-	FA	EN	FA
11	Valli	22	47317	M	L	2M	12	RMP	P0L0	-	-	-	N	-	-	FA	R	UO	F	4	M	-	FA	EN	FA
12	Jaya	32	21056	M	L	1M	12	RMP	P2L2	-	-	-	N	-	-	FA	L	UI	F	2	M	-	FA	EN	FA
13	Saundarya	24	28661	M	L	1M	13	RMP	P1L1	-	-	-	N	-	+	FA	R	UI/LO	S	2	M	-	FA	EN	FA
14	Mariammal	17	22296	U	L	1Y	12	RMP	-	-	-	-	N	-	-	GFA	L	UI	F	6	M	-	FA	EN	GFA
15	Meena	43	16695	M	L+P	3M	12	RMP	P3L3	-	-	-	V	-	-	GFA	L	UO/LO	F	9	M	+	FA	EN	PT + HG
16	Manimegalai	21	11256	U	L	2M	13	RMP	-	-	-	-	N	-	-	FA	L	UO	F	4	M	-	FA	EN	FA
17	Ranjitha begum	40	11281	M	L+P	3M	13	RMP	P2L2A2	-	-	-	N	-	-	GFA	L	UO/UI	V	7	IM	-	FA	EN	FCD
18	Victoria	26	291	M	L	3M	12	RMP	P2L2	-	-	-	N	-	F	FA	L	UO	F	5	M	-	FA	EN	FA
19	Sankareswari	34	59511	M	L	6M	12	RMP	P2L2	-	-	-	N	-	-	FA	L	UO	F	5	M	-	FCD	EX	FA
20	Sasikala	39	39115	M	L+P	2M	12	RMP	P2L2A1	-	-	-	N	-	F	FA	L	LO	F	3	M	-	FA	EN	FA
21	Iyyammal	43	10634	M	L+P	4M	14	RMP	P2L2	-	-	-	N	-	-	DUCT PAPILLOMA	L	C	F	4	M	-	FA	EN	FA
22	Mallika	34	51217	U	L+P	3M	13	RMP	-	-	-	-	N	-	-	FA	L	UO	F	4	M	-	FA	EN	FA
23	Vellathai	17	2540	U	L+P	20D	11	RMP	-	-	-	-	N	-	-	FAD	R	LO	F	3	IM	-	FA	EX	FA
24	Esakipriya	17	14234	U	L+P	2M	12	RMP	-	-	-	-	N	-	-	FA	B/L	LI/UO	F	2.5/5	M	-	FA	EN	FA
25	Esakiammal	13	46702	U	L+P	M20D			-	-	-	-	N	-	-	FA	R	UO/LO	V	7.5	M	-	FA	EX	BENIGN PHYLLOIDS
26	Selvi	40	46117	M	L+P	1 1/2Y	11	RMP	P0L0	-	-	-	N	-	-	FA	R	UO	F	2	M	-	FA	EN	BENIGN PHYLLOIDS
27	Mariammal	48	45531	M	L+P	8M	12	RMP	P3L0	-	-	-	N	-	-	FAD	L	UO	F	5	IM	-	FCD	EX	FA
28	Sivagami	32	1667	M	L+P	11M	14	IMP	P2L2	-	-	+	N	-	-	FA	R	UO	F	3	M	-	FA	EX	FCD
29	Annapackiyam	28	7091	M	L+P	2M	13	IMP	P1L1	-	-	-	N	+	-	FA	L	UO	S	4	M	-	FA	EN	FA
30	Mydeen Fathima	45	14891	M	L+P	11M	11	RMP	P4L4	-	-	+	N	-	-	FA	L	UO	S	5	M	-	FA	EN	BENIGN PHYLLOIDS
31	Sulaimaan	30	16016	M	L+P	3Y	13	RMP	P2L2	-	-	-	N	-	-	FA	R	UO	F	4	M	-	FA	EN	FA
32	Padmavathy	24	17452	U	L	1 1/2Y	12	RMP	-	-	-	-	N	-	-	FA	L	UO	F	4	M	-	FA	EN	FA
33	Mutheswari	33	4530	M	L	9M	11	RMP	P2L2	-	-	-	N	-	-	GFA	R	LO	F	6	M	-	FA	EX	LG-P
34	Vellammal	40	407333	M	L+P	1Y	11	IMP	P3L3	-	-	-	N	-	-	FA	R	UI	F	4	M	-	FA	EN	FA
35	Aruna	32	33052	M	L+P	1Y	12	RMP	P2L2	-	-	-	N	-	-	FA	R	LI	F	2	M	-	FA	EX	FA
36	Subbulakshmi	20	9287	U	L+P	20D	12	RMP	-	-	-	-	N	-	-	FA	L	UI	F	2	M	-	FA	EN	FCD
37	Prema	28	23651	M	L	5M	12	RMP	P2L2	-	-	-	N	-	-	FA	R	LO	F	5	M	-	FA	EN	PHYLLOIDS LG
38	Suganya	19	32381	U	L	20D	13	RMP	-	-	-	-	N	-	-	FA	R	UO	F	3	M	-	FA	EN	TA
39	Rajammal	50	28711	U	L+P	20D	13	IMP	P4L4	-	+	-	N	+	-	P	L	UO/IO	V	14	IM	+	P	WLE	BODERLINE P
40	Sanovar	25	30405	M	L	3M	11	RMP	P2L3	-	-	+	N	-	-	GFA	L	UO	H	7	M	-	FA	EN	PERICAN GFA
41	Rajalakshmi	42	28649	M	L+P	4M	12	RMP	P3L2D1	-	-	-	N	-	-	GFA	R	UO/IO	F	6	M	-	FA	EN	FA
42	Arumaithai	55	28273	M	L+P	11M	13	RMP	P4L3	-	+	-	N	-	-	GFA	L	LO/LI	F	6	M	-	FA	EN	P-LG
43	Sathya	20	22720	U	L+	3M	12	RMP	-	-	-	-	N	-	-	FA	L	UO	F	4	-	-	FA	EN	FA
44	Sivarani	44	22495	M	L+P	7M	12	RMP	P2L2	-	-	+	N	-	-	PHYLLOID	R	UO/IO	F	10	M	+	PHYLLOIDS	WLE	P-LG
45	Muthulakshmi	35	14251	M	L+P	8M	12	RMP	P3L3	-	-	+	N	-	-	FA	L	UO/IO	F	5	M	-	FA	EN	FA + OSSEOUS METAPLASIA
46	Pitchirose	21	19294	U	L	25D	12	RMP	-	-	-	-	N	-	-	FA	L	UO	F	3	IM	-	FA	EX	TA + FOCAL EPITHELIOSIS
47	Jhansi	20	19792	U	L+P	1M	13	RMP	-	-	-	-	N	-	-	GFA	L	UO/UI	F	8	M	-	FA	EN	TA
48	Gnanammal	50	17281	M	L+P	2M	13	RMP	-	-	+	-	N	-	-	P	R	UO/IO	F	7	M	-	P	WLE	P-IG
49	Saundarakumari	22	8281	U	L	20D	13	RMP	-	-	-	-	N	-	-	FA	L	UO	F	1	M	-	FA	EN	TA

50	Mariammal	18	1424	U	L	20D	12	RMP	-	-	-	-	N	-	-	FA	L	UO	F	5	M	-	FA	EN	FA
51	Maheswari	18	14594	U	L+P	3M	13	RMP	-	-	-	-	N	-	-	FAD	L	UO	F	5	M	-	FA	EX	FA
52	Amudha	36	14023	M	L	20D	14	RMP	P2L2	-	-	-	N	-	-	GFA	L	LO	F	7	IM	-	FE/DH	WLE	LGP
53	Saraswathi	50	13005	M	L+P	2M	13	RMP	P2L2A1	-	+	-	N	-	-	GFA	R	UO	F	8	M	-	FA	EN	LGP
54	Deepa	42	52642	M	L+P	6M	13	RMP	P2L2	-	-	-	N	-	-	FCD	L	UI	V	5	IM	-	FA	EX	FA
55	Muthulakshmi	33	8239	M	L	11M	13	RMP	P2L2	-	-	-	V	-	-	FA	R	UO	F	4	M	-	FA	EN	FA
56	Chandra	25	51553	U	L	2M	12	RMP	-	-	-	-	N	-	-	FA	L	UI	S	4	M	-	FA	EN	FA
57	Anthayammal	33	23654	M	L+P	1Y	13	RMP	P2L2	-	-	+	N	-	-	FAD	R	UO	S	3	IM	-	FE/DH	WLE	TA
58	Parveen	15	7485	U	L+P	1M	12	IMP	-	-	-	-	N	-	-	GFA	R	UO/UL	H	9	M	+	FA	EN	GFA
59	Marijeya	38	17043	M	L	3M	12	RMP	P2L2	-	-	-	N	-	-	GFA	L	UO/UL	F	7	M	-	FA	EN	GFA
60	Joshi	20	197112	U	L+P	2M	14	RMP	-	-	-	-	N	-	F	FA	L	LI	F	3	M	-	FA	EN	FA
61	Thangapushpam	45	22329	M	L	2M	13	RMP	P2L2	-	-	-	V	-	-	FA	L	LO	F	3	M	-	FA	EN	FA
62	Murugalakshmi	34	5889	M	L+P	11/2Y	12	RMP	P2L2	-	-	-	N	-	-	FA	L	UO	F	5	M	-	FA	EN	FA+DUCT EPITHELIOSIS
63	Saranya	1+9	32334	U	L	20D	13	RMP	-	-	-	-	N	-	-	FA	R	UO	F	4	M	-	FA	EN	FA
64	Thangalakshmi	29	37891	U	L	20D	13	IMP	-	-	-	-	N	-	-	FA	L	UO	F	5	M	-	FA	EN	FA
65	Kasthuribhai	21	31314	U	L+P	4M	11	RMP	-	-	-	-	N	-	-	FA	L	UO	F	3	M	-	FA	EN	FA
66	Rajalakshmi	35	22435	M	L	3M	12	RMP	P2L2	-	-	-	N	-	-	FA	R	LO	F	3X2	IM	-	FA	EN	FCD
67	Chitra	27	22473	M	L+P	20D	11	IMP	P2L2	-	-	-	N	-	-	FA	R	UO	F	5	M	-	FA	EN	FA
68	Utchimahali	27	50690	M	L	25D	14	RMP	P1L1	-	-	-	N	-	-	FA	R	UO	F	4	M	-	FA	EN	FA
69	Jeya	34	54668	M	L+P	1Y	11	RMP	P4L4	-	-	-	N	-	F	FA	B/L	UO/LI	F	4X5	M	-	FA	EN	FA
70	Sivagami	43	54714	M	L+P	4M	11	RMP	P4L4	-	-	-	V	-	-	FA	L	UO	V	4	IM	-	FCD	EX	FA
71	Valliammal	47	17182	M	L+P	3M	12	RMP	P3L3	-	-	-	N	-	+	P	R	UO/LO	V	10	M	-	P	WLE	P-IG
72	Vijaya	22	59656	M	L	30D	13	RMP	P2L2	-	-	+	N	-	-	FA	R	UO	F	4	M	-	FA	EN	FA
73	Muthuarasi	39	23652	M	L+P	6M	12	IMP	P3L3	-	-	-	N	ON R SIDE	-	FA	L	UI	F	3	M	-	FA	EN	FA
74	Shanthi	29	22840	M	L+P	2M	13	IMP	P2L2	-	-	-	V	-	-	FA	R	UO	F	3	M	-	FA	EX	FA
75	Latha	33	35296	M	L	5M	13	RMP	P2L2	-	-	-	N	-	-	FA	L	LO	F	5	M	-	FA	EN	FA+FDE
76	Booma	20	33890	U	L+P	1M	14	RMP	-	-	-	-	N	-	-	FA	R	LO	F	3	M	-	FA	EN	FA
77	Rani	27	47064	M	L	2Y	12	RMP	P1L1	-	-	-	N	-	-	FA	L	UO	F	2	M	-	FA	EN	FA
78	Saraswathy	50	13005	M	L	2M	12	RMP	P6L6	-	+	-	N	-	-	FA	L	UO	F	5	M	-	FA	EN	FA
79	Muthulakshmi	18	47067	U	L	3M	11	RMP	-	-	-	-	N	-	-	FA	B/L	UO/UI	F	4X3	M	-	FA	EN	FA
80	Muthulakshmi	18	21254	U	L+P	3M	14	RMP	-	-	-	-	N	-	-	FA	R	UO	F	4	M	-	FA	EN	FA
81	Sathya	20	22720	U	L+P	2M	13	RMP	-	-	-	-	N	B/L SIDE +	+	FA	L	UO	F	3	M	-	FA	EN	FA
82	Usha	17	22731	U	L	1M	11	RMP	-	-	-	-	N	-	-	FA	L	UO	F	4	Ax N+/M	-	FA	EN	FCD
83	Lakshmi	30	32641	U	L+P	2Y	14	RMP	-	-	-	-	N	-	-	FA	R	UO	F	1	M	-	FA	EN	FA
84	Kanagarathi	30	21257	M	L+P	3M	11	RMP	P2L3	-	-	-	N	-	-	FAD	R	UO	F	2	M	-	FA	EN	FA
85	Essakiammal	19	7439	U	L	2M	12	RMP	-	-	-	-	N	-	-	FA	L	UI	F	3	M	-	FA	EN	FA
86	Susaimuthu	65	39738	M	L	1M	13	RMP	P6L5	-	-	-	N	-	-	FA	L	C	F	5	M	-	FA	EN	FA
87	Murugeswari	21	82884	U	L	2M	12	RMP	-	-	-	-	N	-	-	FA	L	UO	F	4	M	-	FA	EN	FA
88	Umajeyashree	21	68908	U	L	1M	14	IMP	-	-	-	-	N	-	-	FA	R	UO/C	F	7	M	-	FA	EX	GFA
89	Saral	20	10807	U	L+P	20D	14	RMP	-	-	-	-	N	-	-	FA	L	UO	H	8	M	-	FA	EN	GFA
90	Parvathy	39	25489	U	L+P	1M	11	RMP	-	-	-	-	N	-	-	GFA	L	UO/C	S	4	IM	-	FA	EN	FA+FCD
91	Maheswari	18	13665	U	L	2M	13	RMP	-	-	-	-	N	-	-	FA/BR. ABS	L	UO	V	7	M	-	FA	EN	LGPT
92	Petchiammal	23	11253	M	L+P	2M	12	RMP	P1L1	-	-	-	N	-	-	FA	L	UI	V	3	M	-	FA	EN	FA
93	Janaki	30	30989	M	L	1M	12	RMP	P3L3	-	-	-	V	-	F	FA	R	UO	F	4	M	-	FA	EN	FA
94	Thirumalaiselvi	22	29556	U	L	2M	13	RMP	-	-	-	-	N	-	-	FA	L	LI	F	4	M	-	FA	EN	FA
95	Pushpalatha	37	3472	M	L+P	3M	14	RMP	P3L3	-	-	+	N	-	-	FA	L	UI	F	3	M	-	FA	EN	FA
96	Kanchana	44	9228	M	L+P	6M	14	RMP	P4L4	-	-	-	N	-	-	FA	R	UO	F	2	IM	-	PITHELOS	EX	EPITHELIOSIS
97	Subulakshmi	35	38574	M	L+P	2Y	11	IMP	P3L3	-	-	-	N	-	-	FA	B/L	UO/IO	V	4X3/3X2	IM	-	FCD	EX	FA
98	Sarojini	24	31726	U	L	3M	11	IMP	-	-	-	-	N	-	-	FA	R	C	F	7	M	-	FA	EN	GFA
99	Theresal	30	58802	M	L	1M	14	RMP	P3L3	-	-	-	N	-	-	FAD	L	C/UO	F	12	M	+	FA	EN	GFA
100	Vijayammal	48	34053	M	L	1Y	11	RMP	P3L3	-	+	-	N	-	+	GFA	R	UI	F	4	M	-	FA	EN	FA
101	Zeelavathy	39	29917	M	L+P	1M	13	RMP	P2L3	-	-	-	N	-	-	FA	R	UO	V	4	IM+LN	-	FCD	EX	P-LG
102	Dharshini	17	40496	U	L	20D	13	IMP	-	-	-	-	N	-	-	FA	L	UO	F	3	M	-	FA	EN	FA
103	Maragathavalli	52	37589	M	L	6M	14	IMP	P6L4	-	+	-	N	-	-	FA	R	UO	F	5	M	-	FA	EN	FA
104	Shalini	27	38789	M	L+P	10D	11	RMP	P2L2	-	-	-	N	-	-	FA	R	LI	F	4	M	-	FA	EN	FA
105	Subbulakshmi	22	1872	M	L+P	20D	13	RMP	P1L1	+	-	-	N	-	-	FA	L	UO	V	8X5	IM	-	FA	EN	FA
106	Peratchi	47	8802	M	L+P	15D	12	IMP	P3L3	-	-	-	N	-	-	FA	L	UO/C	V	12X8	M	+	FA	EN	FA
107	Pappa	40	32851	M	L+P	20D	11	RMP	P2L2	-	-	-	N	-	-	BR. ABS	L	C/LO	F	5X7	M	+	FA	EN	FA
108	Chinnathai	40	3448	M	L+P	25D	13	RMP	P2L2	-	-	-	N	-	-	BR. ABS	R	C/UO	F	6X5	M	+	FA	EN	FA
109	Ponmuthai	24	3185	M	L+P	10D	12	RMP	P1L1L	-	-	-	N	-	-	BR. ABS	R	C/UO	F	6X7	M	-	FA	EN	FA